THE LARYNGOSCOPE

AN INTERNATIONAL MONTHLY JOURNAL
ON DISEASES OF THE

EAR-NOSE-THROAT

FOUNDED IN 1896 BY
DR. M. A. GOLDSTEIN, ST. LOUIS

VOLUME LXVIII—January-December, 1958.

DR. THEO. E. WALSH, ST. LOUIS
EDITOR

PUBLISHED BY THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY - ST. LOUIS (10), MO., U.S.A.

THE
LARYNGOSCOPE.

VOL. LXVIII JANUARY, 1958 No. 1

EXPERIENCES IN THE TREATMENT OF THE ALLERGIC NASAL POLYP BY THE INTRAPOLYP INJECTION OF PREDNISOLONE T. B. A.

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Historically, the presence of the nasal polyp has been known for centuries. It is stated that Hippocrates described nasal polypi, gave theories as to their origin, and described techniques for their removal. Many theories of polyp formation have been described from these early days.

The pioneer and monumental works of Drs. Kern and Schenck[1,2,3,4,5] helped to establish, without doubt, the allergic origin of the nasal polyp. Other pioneers in the description of the allergic origin of the nasal polyp were Hansel,[6,7] Weille,[8] Coates and Ersner.[9]

Histopathologic study indicated the intense infiltration and edema of the tunica propria. The most recent advances have imparted further information as to the importance of connective tissue and the so-called ground substance. The diseases producing the changes in the ground substance have been called by Klemperer[10]—the collagen diseases. Semenov[11] believes that the nasal polyp is a manifestation of the collagen

*Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 6, 1957.

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†The prednisolone® T.B.A. was supplied by Merck Sharp and Dohme—Brand Hydeltra T.B.A.

‡The study on the pathology of the polyp was carried out by Dr. Elizabeth Holmes of the Department of Pathology, Temple University Medical Center, under a grant supplied by the Sharp & Dohme Co. Further publications of these studies are to follow.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication July 3, 1957.

disease. The pathologic changes in almost every case center in the mesenchymal substance of the tunica propria. Rawlin[12] has suggested that the nasal polyp resembles embryonal, mesenchymal tissue. This recent experimental and histologic study, which is too extensive to mention in this short presentation, establishes that the nasal polyp is allergic in origin, and that the pathologic change is in the subepithelial tissue or connective stroma. This is due to some abnormality of the connective tissue stroma and of the ground substances. The polyp is not a true tumor, but is a swollen mass of thickened connective tissue. Proof exists that the hormonal effect of ACTH and Cortisone affects more directly the ground substance of the connective tissue.

Bordley,[13,14,15] and his associates at the Johns Hopkins Hospital, were among the first to notice the amazing changes that occurred in the mucosa of the upper respiratory tract. They noted the fact that nasal polypi completely or almost completely disappeared. Other reports soon followed. Thorn et al.[16] noted shrinkage, but not complete regression of polypoid and antral mucous membrane, following the use of ACTH in allergic states. As occurs after the discovery or availability of any new drug or substance, it is subjected to a general trial by every field. As the result of the experiences in other fields, many uses of ACTH and Cortisone have been started in otolaryngology.

Ashley[17] treated six patients with chronic allergic rhinitis and polypoid nasal mucosa, and found a striking change in the tissues of the nose and pharynx. Semenov[11] treated patients with polypoid rhinitis, with topical Cortisone and by injection of Cortisone into the septal mucosa and polyp. Davison[18] suggested the treatment of hyperplastic sinusitis of allergic origin with bacteriocidal doses of penicillin and Cortisone orally. Dill and Bolstad[19] used Cortisone locally in two reports. Wall and Shure[20] used the injection of Cortisone submucosally in the inferior turbinate. Randolph and Rollins[21] used ACTH locally in clinical ragweed hay fever and asthma.

Stewart and Kawa[22] reported experiences with Cortisone and ACTH in the treatment of allergic rhinitis. Smith[23]

reported on the local use of hydrocortone acetate in the nose. Other reports on the intranasal use of medication containing Cortisone were made by Seidman and Schaffer,[24] Schwarz,[25] Silcox[26] and Evans.[27] Hotchkiss[28] reported on the oral use of prednisolone in nasal polyposis. Owen[29] reported on the treatment of nasal polyposis by the injection of hydrocortisone directly into the nasal polyp.

The purpose of this paper is to report our own experiences with the treatment of nasal polypi by the intrapolyp injection of corticoid substances. This has extended over a period of six years. This included the injection of ACTH, Cortisone, hydrocortisone, hydrocortisone T.B.A., and during the past one and one-half years, with Prednisolone T.B.A. (in the form of suspension Hydeltra® T.B.A., supplied by Sharp & Dohme, Division of Merck & Company.

We first used ACTH and Cortisone approximately six years ago, after reading the reports of the first ACTH conference and the experience of Bordley, Hench, and others previously quoted. Our first experience was with ACTH. The first patient was a man, age 60. He was a baker, and gave a history of allergy of many years' duration. He had already undergone numerous bilateral polypectomies, and several radical sinus procedures. After several intrapolyp injections of ACTH, the polypi disappeared completely, and to date have not recurred. We then treated three or four other patients similarly and found a regression of the polypi.

Cortisone then became available, and it appeared to us that since ACTH acted on the adrenal glands to produce Cortisone, that using Cortisone itself locally, might produce a more beneficial, and a quicker result. It is noted that when using the intrapolyp injection of Cortisone solution, poor results were obtained. After treating eight patients in this manner, it appeared that this treatment was not beneficial. Soon after, in December, 1951, the experiences of Hollander[30] and his coworkers, using intra-articular injections of Hydrocortisone (or Compound F) were reported. This report indicated that this was a safe method of injection and treatment, and resulted in local relief of symptoms. It was then that we began to use this particular drug in the intra-polyp injection ther-

apy. It was found that there was a prompt regression of the nasal polyp with the injection of Hydrocortisone solution into the polyp. In this time, a total of 38 patients have been treated by the intrapolyp injection of Hydrocortisone solution T.B.A., with a total of 228 injections. At no time was there a local or systemic reaction following this injection.

In May, 1956, we began to use Prednisolone T.B.A. in the form of suspension Hydeltra®T.B.A., supplied by Sharp & Dohme Company. This is Prednisolone tertiary-butylactate (Merck), a very slightly soluble ester of Prednisolone, and it is capable of producing longer local anti-inflammatory action. This report will concern itself with our experiences with Prednisolone T.B.A., since others mentioned above have reported on the use of the other compounds.

Since we started using this compound, we have treated 30 patients with nasal polyposis by the injection of Hydeltra® T.B.A. The patients were given over 150 intranasal injections, and there were no untoward reactions, either locally or systemically.

The following is a description of the technique: Approximately 2 cc. of solution are used at each treatment. One cubic centimeter is injected into the polypi of each nasal chamber. Each cubic centimeter contains 20 mgm. of Prednisolone tertiary butyl acetate. The material is injected into the polyp in the following manner: The nasal membrane is sprayed with a solution of one per cent ephedrine sulfate. This renders the nasal polypi relatively more accessible. The actual injection into the polyp is done without local anesthesia, since the polyp or polypoid tissue seems to be insensitive to the entrance of the needle. A 25-gauge, 3½-inch needle is used. The needle is inserted into the body of the polyp, and a few drops of the Prednisolone are injected, until the back pressure on the piston of the syringe is felt. This is repeated from polyp to polyp until 1.0 cc. is given in each nasal chamber. The total dose of Hydeltra® T.B.A. is about 30 to 40 mgm. at each treatment, depending upon the amount of solution the polyp will accept. This treatment is repeated each five to seven days, see Figs. 1 and 1-A.

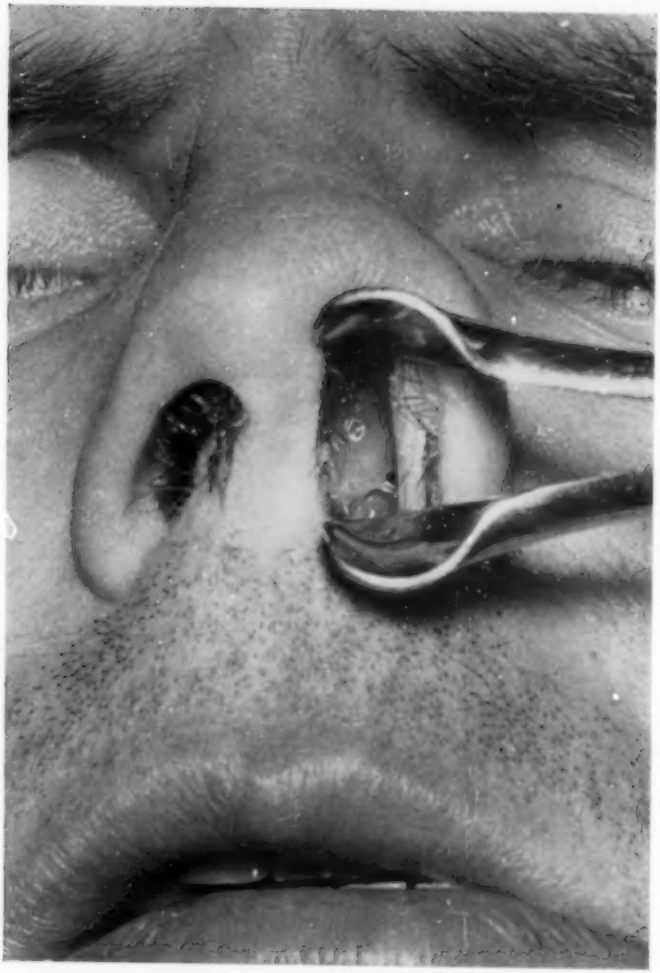

Fig. 1. A typical nasal poyp.

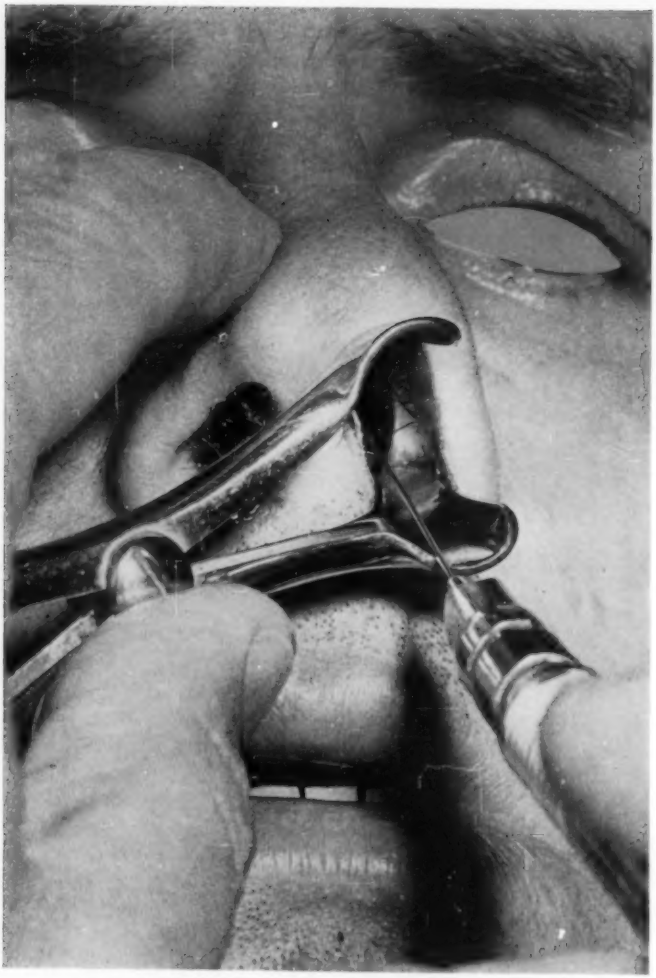

Fig. 1-A. Technique of injection of the nasal polypi.

Following the first or second injection, the nasal mucosa becomes a pale, bluish white. The edema subsides. The polyp will often regress more than half during this time. Sometimes after three or four injections, the polyps are practically gone. The patients state that they had not only relief of breathing, but many of them felt that they had regained their sense of smell. During the course of this investigation, biopsies were taken prior to the injection, and a study of the polyp was made. On several occasions, a biopsy was taken immediately after the Hydeltra® T.B.A. was introduced into the polyp, but it was found that microscopically no evidence of the presence of the medication could be seen. As the polyps regressed in size, further biopsies were taken. It was noted that as long as there was any polyp tissue present, despite its decreased size, the histologic appearance varied little from the full sized polyp.

Experiences from pathologic studies—When this study was started, and sections of polyp tissue were taken, it was hoped that histologic study would reveal noteworthy changes. It was noted, however, that despite the regression of the size of the polyp, as long as any polypoid tissue remained, the histologic picture was identical. In collagen diseases, however, microscopic changes are difficult to note. It is probably necessary to view the tissue with the electron microscope to note changes in the fibrillary structure. More important is the probability that the changes that occur, which result in the production of the polyp, are in the ground substance of the tunica propria, and are chemical changes rather than histologic changes.

The following are excerpts from the histopathologic reports of nasal polypi of several patients under treatment with the intrapolyp injection of Prednisolone:

Case 1. Sections, labeled from right and left nasal chambers, show a similar picture. The sections show polypoid tissue partially covered by pseudostratified columnar epithelium. The stroma is extremely loose and edematous, and is liberally sprinkled by leukocytes, which are predominantly plasma cells and eosinophils.

There are areas of increased capillarity and of catarrhal activity of contained mucus glands. The former feature is present beneath the surface mucosa, thus suggesting granulation tissue formation. The edema and the eosinophilic response would support the clinic impression that these polyps are on an allergic basis.

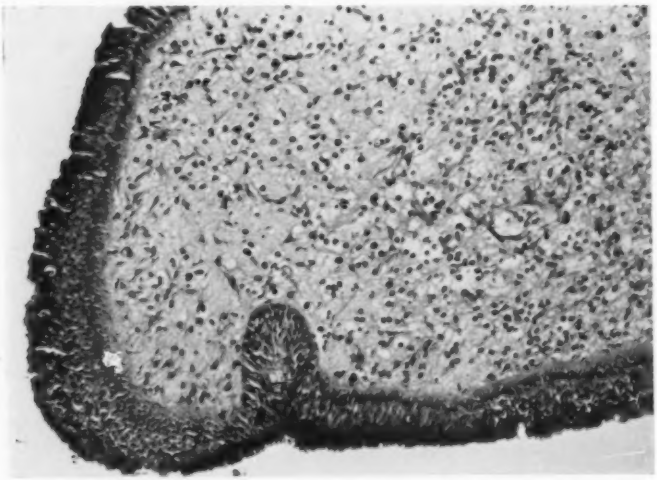

Fig. 2. Nasal polyp after one injection of Prednisolone T.B.A.

After an injection of Prednisolone, the polyp was reduced to approximately half its size on gross inspection. A biopsy was taken which revealed the following, see Fig. 2. The polypoid structure shows an edematous stroma which contains focal collections and a diffuse of lymphocytes and eosinophils. The surface epithelium is intact and of the tall columnar, occasionally pseudostratified type.

Case 2. A nasal polyp after five injections of Prednisolone T.B.A. The nasal polyp described here was very small. On gross inspection of the nose it had almost disappeared compared to its appearance when the injection treatment was started. It was noted histologically that the essential struc-

ture remains unchanged, with the exception of the regression of the edema, see Fig. 3.

The polypoid fragments show considerable edema, and mild to moderate leukocytic infiltration of the stroma. The The latter response is in the form of small groups of cells in some areas, and in other places the cells have a scattered, sprinkled appearance. These cells are of all varieties with the plasmocyte and the eosinophile predominating. There is

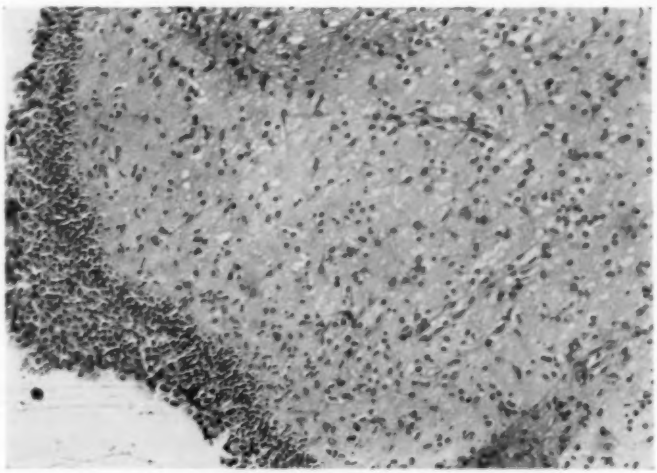

Fig. 3. Nasal polyp after five injections of Prednisolone.

one small area of recent hemorrhage, surrounded by fibroblasts and by pigment-laden cells, but this does not have the appearance of venous thrombosis. The surface epithelium is thickened but of the pseudostratified respiratory tract type.

Other observers have similar experience on histologic study. Rappaport, et al.,[28] state as follows: "The mucosa of 46 patients with ragweed pollenosis were studied before and after ACTH therapy. The mucosa obtained at the end of four days of treatment with ACTH, when compared with that removed before treatment, showed consistently the following

changes: *1.* a general increase in stainability of the glycoprotein ground substance; *2.* an increase in the cement substance between the epithelial cells of the mucosa; *3.* an increase in the staining density and in the width of the submucous and of the perivascular "basement membrane"; *4.* an increase in the number, the thickness and the stainability of the fibers in the ground substance; *5.* an increase in the number and the size of red-staining granules in the cytoplasm of stellate, oval and round cells present in the submucosa. These cells are believed to be undifferentiated fibroblasts (mesenchymal cells), and the granules to be glycoprotein in composition. It would appear from these results that during the allergic reaction the glycoproteins of the ground substances and the basement membranes are broken down (depolymerized) to smaller units with a proportionate loss of stainability. The effect of Corticotropin is to arrest the process of depolymerization. Glycoproteins are restored to a normal state with a resulting increase in stainability.

In several patients the polyp reached a certain point in its regression, and then did not become smaller after further injections. In those it was thought that possibly some fibrotic change in the mucous membrane had occurred, and that the remnants of the polyp could be removed surgically. Some polypi did not reduce in size following the injection, and it was thought that possibly some etiologic factor other than allergy was involved, and that treatment was discontinued. In some patients there was a recurrence after several weeks or several months. This often followed an upper respiratory infection, or after contact with a known allergen, or some psychosomatic upset. In these patients, the injections were repeated, and the same effect was obtained. The injections may be repeated numerous times with the same beneficial results, and apparently without reaction or ill effects.

From my experiences with injections of Prednisolone T.B.A., I found that it was a safe method, and that no local or general reactions have occurred. A regression of the polypi occurs promptly, and the patient has considerable relief. There is no pain or bleeding. This method may be used to give temporary relief while allergic control and man-

agement is obtained. It may be used in patients who are unsuited for surgery because of various physical reasons. It may be used during acute allergic exacerbations while awaiting the benefit of allergy study and desensitization. Kern and Schenck[1] long ago recommended that surgical procedures be avoided during acute allergic exacerbations, such as pollen season, or during acute asthmatic exacerbations. This method will give the patient considerable relief while in this acute stage. Many times when the maximum benefit of injection therapy is reached with Prednisolone T.B.A., the residual polyp tissue may be removed with very little trauma to the nasal mucosa or sinus mucosa. In the event of recurrence, it is a simple matter to repeat the injections and see the same beneficial results. Hollander[30] found that repeated injections in the arthritic are equally effective and may maintain remission of symptoms for long periods. He also found an absence of systemic effects and adverse reactions from this local method of utilizing hydrocortisone. Cessation of injections has not been followed by exacerbations of the local inflammation, but only by relapse to pre-treatment status. In severe allergic states, the remnants of any nasal mucous membrane, however small, can be the site of polyp or polypoid formation; therefore, even if the polyp recurs after the Hydeltra® T.B.A., then the further injection may result in its disappearance, or surgical removal can be carried out.

Many observers have shown that despite the degree of radical surgical treatment there will be recurrence of polypoid rhinitis, polypoid sinusitis, or bronchial asthma, unless some degree of allergic control can be obtained. Kern and Schenck,[1] Tuft and Blumstein,[31] Pepys and Duveen,[32] and Chait[33] have reported regression of nasal polypi after proper and continued allergic management and desensitization therapy. The lesson to be gained is that the nasal polyp and the lining membrane of the paranasal sinuses are capable of return or regression to normal.

Some typical case reports are as follows:

Case 1. The patient, age 47, male, was first seen on May 10, 1956. His history revealed nasal congestion, sneezing, rhinorrhea and headache. Twelve years ago an intranasal polypectomy had been performed, and again three years ago. The present examination revealed multiple nasal

polypi in each nasal chamber. The polypi were injected with Predisolone T.B.A. Three treatments were given at weekly intervals. At each examination it was noted the polypi were smaller, one week after the last treatment they were no longer visible. The patient has remained comfortable since this treatment.

Case 2. This patient, a female, was first seen in October, 1946. The patient stated that during the past year she had developed rhinorrhea, cough, asthma and sneezing. She lost her sense of smell and taste. In November, 1945, a polyp had been removed from each nasal chamber. At this examination, the nasal mucosa was pale and boggy. The right middle turbinate was hypertrophied. Many large polypi were present in the right middle meatus. On transillumination, both antra were cloudy. Under local anesthesia the nasal polypi were removed. The patient was next seen in December, 1948, at which time she stated the polypi had recurred within a short time, and they were again removed by another rhinologist. The patient then had a severe attack of asthma and was hospitalized successively at four different hospitals by as many physicians. The nasal examination now revealed multiple nasal polypi bilaterally, which completely occluded each nasal chamber. It was suggested that complete allergic care and management was indicated. Sinus X-rays were made December 6, 1948, at Temple University Hospital where it was shown that both maxillary sinuses were completely opaque. Despite continued desensitization therapy, medical care and management, the patient's asthma continued and her polypi remained. A bilateral transantral ethmoidectomy was done in February, 1949. In January, 1950, polypi were again removed. In January, 1951, when she was again seen, ACTH was available and it was then that the injection of the nasal polypi with ACTH was done. The patient returned for a series of injections into each nasal chamber. The polypi became smaller after each injection, and she began to feel more comfortable. Cortisone then became available and on April 28, May 15, and May 28, 1951, Cortisone was injected into the polypi. This did not reduce the size of the polypi. Because of the poor results with Cortisone, ACTH was again injected and this time after several injections, the polypi disappeared. On February 16, 1953, both nasal chambers were free of polypi. The patient returned again in January, 1956, polpi were again present, and this time the Prednisolone T.B.A. was used. After four treatments both nasal chambers were clear. This case illustrates that surgery alone cannot control nasal polyposis. The cure is bound up in the general allergic state, and until this can be completely managed polypi will recur. The intrapolyp injection is a mode of management and helps avoid repeated surgery.

Case 3. This patient, male, age 53, was first seen with a chief complaint of nasal congestion, rhinorrhea and sneezing. He had had four previous polypectomies. The present study revealed recurrence of polypi with complete closure of both nares. Prednisolone T.B.A. was injected into the polypi. The injections were repeated at weekly intervals, and it took seven treatments to free the nose of polypi. There were no untoward local or systemic reactions. When last seen the nose was free of polypi.

Case 4. A female patient, age 40, was seen on May 18, 1956, with a history of asthma for the past four years. Four previous operations for the removal of nasal polypi had been done, and she still complained of nasal congestions and discharge. Examination revealed recurrent bilateral polyposis and allergic rhinitis. The polypi were injected with Hydeltra T.B.A. on eight occasions. These injections were made at weekly intervals. After the fifth injection, the polyps were practically gone except for small residual polypoid areas. On the left side a dense fibrous sac, the residual of one polyp, remained. This sac was removed with a nasal snare. The patient was re-examined in two months at which

time the nose was still clear. The patient was referred for general allergic study and management. At present she is receiving desensitization therapy.

Case 5. R.F., age 42, was first seen in December, 1946, at which time a bilateral polypectomy was performed. He was well then until December, 1955, at which time he was admitted to the Temple University Medical Center with asthma and hypertension, and anginal symptoms. Both nasal chambers were completely filled with nasal polypi. These nasal polypi projected from the nares (see Fig. 1). Daily injections of Prednisolone Hydeltra T.B.A. were given for a five day period, following which the complete regression of the nasal polypi was noted.

The injection technique helps with the management of the allergic patient as illustrated by the following: Mrs. R.P. had allergic rhinitis for many years. Despite constant allergic care and management by an allergist, she had frequent recurrences of nasal polypi. The patient can tell when her nose is obstructed and reports for treatment on her own, at which

SUMMARY OF CASES TREATED.

	Patients	Injections
ACTH	5	28
Cortone	5	40
Cortone F (Hydrocortisone T.B.A.)	38	228
Prednisolone (Hydeltra T.B.A.)	30	220

time the nasal polypi are injected. The swelling and polypi are reduced, and the patient feels better for varying lengths of time; then she is again seen. In this way, we avoid multiple surgical excisions and keep our patient comfortable.

Case 6. Mrs. R. Z., an elderly female, age 70, had allergic rhinitis for many years. She had numerous polypectomies and four years ago had a bilateral Caldwell-Luc and transantral ethmoidectomy. Despite this, nasal polypi continued to recur. There is a definite seasonal cycle. When the patient has nasal congestion and obstruction, she returns for injections with subsequent relief. In three years of observation she has had numerous injections, with relief for varying periods. Polypi continue to recur, but we have avoided surgical excision.

Control studies have been done at varying times, injecting the polypi with distilled water, hyaluronidase, and antihistaminic substances. No results were obtained in our experiences. This was done only once or twice in the patients selected, because it was felt that it was unfair to withhold relief for a longer trial.

Since the advent of the Prednisolone T.B.A. we have used

this exclusively. There have been no systemic reactions. The polypi are injected, and on the return visit, considerable regression is usually noted. Sometimes, the polypi disappear completely after the first injection. At other times, the remnant of the polyp is seen to be covered with a thick, plastic exudate, and when this is removed by the use of suction, the remnant of the polyp can be aspirated and removed in this way. There were very few allergic polypi that did not decrease in size after the injection. Most patients were relieved by the treatment. The number of doses varies. Some patients have complete regression to a normal state, others only very temporary relief. The amount of relief varies with the allergic problem and the allergic reaction that caused the flare-up and recurrence. In most cases, the regression of the polypi can be hastened by the use of corticosteroid substances in conjunction with the injection therapy. In some patients the decrease in the size of the polyp stops at a certain point and no further improvement can be obtained. In these patients, the conventional snare and punch technique is used for the further removal. Since we have been using this treatment we have done very few surgical polypectomies. This is reserved for the occasional patient who has residual fibrotic tissue after the injections are completed. Most patients who have polyps have already experienced a great deal of surgery, and are happy to have a try at injection therapy even if it means repeated visits.

This treatment would indicate that polypi can be reduced without surgery, and that we can perform a minimal of intranasal surgery. Sometimes on the first visit, if a patient is very uncomfortable, the larger polypi can be removed by the snare, and this gives quicker relief. Then the remaining polypi are treated by injection therapy. This is mentioned to indicate that the steroid injection therapy has a place in our therapy, and its use can be varied by the rhinologist's judgment and experience with the method. In the beginning, the injection was used alone to study its benefit. At times, a combination of the snare, injection and oral corticosteroid therapy give quicker and more permanent relief. In my present experience and thinking, radical attack on the ethmoidal labyrinth will be rarely indicated.

DISCUSSION AND CONCLUSION.

The advent of increased knowledge of the tunica propria, the supporting fibrillar network, and the collagen ground substance, has given an impetus to the study of many types of allergic and inflammatory reactions. The mesenchymal tissue, long neglected, has now assumed an important place. These advances have been possible because of the electron microscope, advanced biochemical research, and the endocrinologic research, resulting in the availability of the many corticosteroid substances. The use of these substances has enabled us to cause the regression of allergic reactions. Hench[34] stated: "Aside from epinephrine, these are the first hormonal agents with marked anti-allergic properties." The treatment of nasal polypi by the use of Prednisolone T.B.A. is described. Good results, with relief of symptoms, have been obtained in most cases. This occurred without local or systemic reaction. It permits the full utilization of the anti-inflammatory activity of corticosteroids at the tissue level without producing systemic effects. In the event of recurrence, the injections can be repeated many times. If there is no response to this therapy, then surgery can be instituted. The important lesson is not a new treatment for polyposis, but that nasal mucous membrane, having undergone polypoid change, is capable of regression to normal, or near normal, and should be respected rather than exenterated.

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PIETRO CALICETI AWARD.

Dr. Samuel Rosen, of New York City, has been honored with the first Pietro Caliceti Award for his outstanding work in stapes mobilization. He is consulting otolaryngologist at Mount Sinai Hospital.

The Award—a medal, diploma and $1600, was made to Dr. Rosen in Bologna, Italy, December 13, 1957. It was established in memory of Professor Caliceti, director of the University of Bologna, who died in 1951. It is to be given on an international basis every four years for the most significant and published work in otorhinolaryngology.

STAPES MOBILIZATION.

Lasting Results at One and Two Years.*†

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and
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TECHNIQUE.

In 1955 we published the results obtained in the first 63 cases operated upon by stapes mobilization in the Otolaryngological Clinic of the University of Helsinki. The operations were performed by the original Rosen technique, though in most cases with the aid of a binocular Zeiss operating microscope. In 25 cases the improvement in hearing was good; in 12, slight, and in 26 no result was obtained. Very many of the failures were due to crural fracture and persisting fixation of the stapes footplate. Our technique has since been modified to obtain better visualization of the oval window and to facilitate mobilization.

In the spring of 1955, at the meeting of the German Otolaryngological Society, Dr. Y. Meurman presented results obtained with his method: the atticus is opened through an endaural incision, and the corpora of the ossicles exposed; this is followed by removal of the bridge which results in good visual exposure of the oval window region. If the stapes is solidly fixed, the footplate itself is mobilized by breaking the bony connections with thin lancet-pointed knives. Opening of the attic, however, is not required for proper visualization of the oval window; therefore, we later adopted the following technique: an ordinary circular endaural incision is made near the orifice of the external meatus, and from the uppermost point of this incision, another incision is carried for

*Read at the meeting of the Sixth International Congress of Otolaryngology, Washington, D. C., May 5, 1957.

†Prof. Y. Meurman, M.D., Head of the Otolaryngological University Clinic, Helsinki.

This manuscript received in The Laryngoscope Office and accepted for publication May 13, 1957.

some distance upward between the tragus and the helix. The skin of the upper and posterior meatal wall is dissected from the bone down to the drum margin, and the posterior margin of the drum is then freed from its sulcus. In the area where the chorda tympani leaves the facial nerve, some of the bony margin of the tympanum is removed, using a bur so as to allow proper visual exposure of the stapedial tendon, the facial canal, the crura, and most important, the footplate itself. Several variations have been used for the actual mobilization. Initially we often tried, after the foregoing approach, to mobilize the footplate by applying pressure on the neck of the stapes in the direction of the stapes tendon, or by applying pressure to the end of the long process of the incus in an attempt to push the stapes inward. During 1956 we began to

TABLE I.

CASES OF STAPES MOBILIZATION (SHAMBAUGH'S CATEGORIES).

	A	B	C	Total
1954	64	29	8	101
1955	89	34	13	136
1956	94	46	2	142
Total	247	109	23	379

use more and more direct mobilization of the footplate, either by breaking the bony connections between the footplate and the window margins with a lancet knife, or with a needle probe. Ordinarily it has sufficed to mobilize the footplate at the anterior periphery of its attachment to the anterior crus and at the posterior periphery of its attachment to the posterior crus, but occasionally it has been necessary to break connections also at the superior and inferior margins of the footplate. As far as possible we avoid fracturing the crura.

MATERIAL AND INITIAL RESULTS.

In Table I the cases are classified by Shambaugh's criteria and year of operation.

The total number of operated ears was 379. This series includes also remobilizations.

Naturally, the initial result as to the amount of hearing gain differed in these various groups. To obtain a more uniform basis of reference we, therefore, present the initial results in relation to the theoretically obtainable level. The cases were divided into four groups, according to the result achieved. In Group I the improvement rose to the theoretically possible level, or very close to it; postoperatively the loss by air was 0-10 db. greater than the preoperative bone conduction loss after adjustment of the Carhart notch. In Group II the gap between the postoperative air conduction and the preoperative bone conduction curve was 15-20 db.; in Group III 25 db. or more, and in Group IV there was no improvement whatever. The results are shown in Table II.

TABLE II.

PRIMARY RESULTS—COMPARISON OF POSTOPERATIVE AIR-CONDUCTION AND PREOPERATIVE BONE-CONDUCTION.

I. Postoperative AC-loss 0-10 db.>preoperative BC-loss.
II. Postoperative AC-loss 15-20 db.>preoperative BC-loss.
III. Postoperative AC-loss 25- db.>preoperative BC-loss.
IV. No Results.

	1954	1955	1956	Total
I.	12—11.8%	21—15.4%	23—16.2%	56—14.7%
II.	29—28.6%	29—21.3%	64—45.0%	122—32.3%
III.	18—17.8%	22—16.2%	32—22.6%	72—19.0%
IV.	42—41.8%	64—47.1%	23—16.2%	129—34.0%
	101	136	142	379

In the above Table, attention is called to the fact that the proportion of cases in Group I has remained practically unchanged. In spite of changes in the operative technique the percentage of cases with an excellent result has not increased appreciably. In Group II, in which the gain is still good, similar to that obtained by a successful fenestration, a distinct improvement was noted in 1956. It is quite obvious that improved visual exposure and the substitution of direct footplate mobilization for the previous indirect method have caused an increase in the cases of this group; similarly, the proportion of complete failures has been considerably reduced, as compared with the preceding years.

Despite the advances in technique, it was not possible to

avoid crural fractures entirely; however, the adoption of direct footplate mobilization has reduced the importance of fractures. This is evident from Table III, in which the number of cases with fracture is shown in the different groups of hearing improvement.

Notwithstanding fracture, the results as to hearing improvement were good, especially in 1956—16 cases.

TABLE III.

NUMBER OF CASES WITH FRACTURE.

	1954	1955	1956
I.	—	2	3
II.	2	3	13
III.	8	6	5
IV.	32	37	11
Total	42	48	32

LASTING RESULTS.

The second, and most important point to be considered when dealing with the mobilization procedure, is the permanency of the result. The patients in our series were from different parts of Finland, and, unfortunately not nearly all of them could be studied by follow-up examinations; therefore, it is not possible to present any definite percentages illustrating the permanency of the results.

Table IV, however, shows data on the cases in which the hearing subsequently receded.

The above Table shows that in Group I, in which the hearing gain was close to the theoretically possible level, the hearing usually did not deteriorate within the first 100 days after the operation. During a longer period of observation cases with lost improvement occur also in this group. In Groups II and III, on the other hand, the majority of the cases which lost the obtained improvement did so within a comparatively short period. The next table (Table V) shows the percentage of cases with maintained improvement, lost improvement, and with lacking follow-up data in the years studied.

This Table shows that the number of cases in which improvement is lost, increases with the duration of the observation period. A more detailed presentation of the lasting results is impossible, because of the great number of cases in which follow-up examination could not be carried out. The figures in the Table obviously do not do full justice to our results as far as lasting restoration of hearing is concerned.

CONCLUSIONS.

In own experience, success of a mobilization operation is gained primarily by an exposure that allows good visualization

TABLE IV.

IMPAIRMENT TO PREOPERATIVE LEVEL.

	1954			1955			1956		
Days	I	II	III	I	II	III	I	II	III
0-100	_	3	8	_	5	13	_	4	9
100-200	_	3	3	_	1	2	_	5	4
200-300	_	3	1	1	_	_	_	2	_
300-400	2	4	_	1	_	_	_	_	_
400-500	_	_	_	1	1	_			
500-600	_	_	_	_	_	_			
600-700	_	1	_						
700	3	_	_						
Total	5	14	12	3	7	15	_	11	13
Total No. of ears operated	12	29	18	21	29	22	23	64	32

of the region of the oval window. It is only by watching the movement of the stapes footplate that the completeness of mobilization can be appraised. The fact that a corresponding movement is seen in the membrane or niche of the round window does not yet insure that the footplate has been fully freed. The stapes footplate must be readily and freely moveable in all possible directions on gentle manipulation.

Sometimes the fixation of the stapedial footplate is very slight. Mobilization is then easy, and can be successfully performed with almost any technique. If, however, the footplate is rigidly fixed, we consider a procedure directed at the footplate itself, the only correct one. Fixation is usually most solid at the anterior periphery of the attachment of the

footplate to the anterior crus, but frequently the footplate is fixed also posteriorly. By applying pressures on these two points, it is possible to mobilize the footplate. Rarely is it necessary to free the footplate along its superior and inferior margins.

Advances in operative technique create an increase in the number of good initial results, as indicated by our figures for 1954, 1955, and 1956. In 1956 the initial result was good in about 60 per cent of the cases, and in only 17 per cent was there no improvement whatever. On the other hand, it should

TABLE V.

PERMANENCY OF RESULTS (Up to March, 1957).

	1954	1955	1956
Improvement lost	31—52.5%	29—40.2%	24—20.2%
No follow-up data	21—35.6%	22—31.6%	52—43.7%
Maintained improvement	7—11.9%	21—28.1%	43—36.1%
	59	72	119

be noted that a result corresponding to the theoretically possible level was obtained in only about 16 per cent, in which footplate mobilization can thus be considered to have been complete. In 44 per cent the result was good, though it did not reach the theoretically possible level. In some of the cases this was due to fracture of a crus, both crura, which reduces the effective movements of the footplate in spite of its mobilization. In some cases, however, the footplate evidently had not been completely mobilized.

The fact that in such cases mobilization may have been incomplete, is supported by the observation that this group, in which the initial result was good but did not reach the theoretically possible level, included cases receding to the preoperative level within a very short period of observation. In the group with the best initial result the hearing gain was maintained much longer, and if hearing subsequently receded, deterioration began much later. *From this it is apparent that not even the best primary result insures a lasting restoration of hearing;* but it would seem that the more incomplete

the mobilization, the more rapidly and frequently does the hearing return to the preoperative level.

We still consider the stapes mobilization operation an acceptable surgical procedure worth developing further, and we regularly perform it first when operating on patients with clinical otosclerosis. If no result is obtained, we then resort to fenestration.

Haartmanink 4.

SEVENTH INTERNATIONAL CONGRESS OF BRONCHOESOPHAGOLOGY.

The Seventh International Congress of Bronchoesophagology will be held at Kyoto (Japan) University, September 12-14, 1958, under the direction of Prof. Mituharu Goto, M.D., Otorhinolaryngological Clinic, Kyoto University Hospital, Kyoto, Japan.

A registration fee of $25.00 U.S.A. will be charged all physicians and a fee of $10.00 for wives or non-medical guests. Registered members are admitted free to banquet, receptions and sightseeing tours.

Closing date for scientific papers will be April 30, 1958. A summary of paper, not to exceed 800 words, should be sent to the Director not later than that date.

GENERAL ANESTHESIA IN BRONCHOSCOPY, ESOPHAGOSCOPY AND LARYNGEAL SURGERY EMPLOYING THE EMERSON "RAINCOAT" RESPIRATOR.*

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Because of the many difficulties associated with general anesthesia when employed for peroral endoscopy, local analgesia, although not necessarily the patient's choice, has remained the endoscopist's choice in these procedures.

There have been numerous general anesthesia techniques described,[1,2,3,4] which have sought to resolve the major problem of maintaining adequate respiratory exchange in a deeply anesthetized or curarized patient, with the surgeon working in the zone of anesthesia administration. All of these methods have the basic physiologic fault of inadequate ventilation with retention of carbon dioxide.

This problem has now been resolved by the use of a modified polio chest respirator, which efficiently maintains respiratory exchange in a lightly anesthetized but totally curarized patient.

The respirator consists of a plastic or rubber raincoat, which slips over the patient's head and trunk. It fits snugly around the neck and wrists. A wire cage is placed under the raincoat so that there is at least a six-inch space between the

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication August 12, 1957.

raincoat and the patient's chest (see Fig. 1). The raincoat is then tucked in snugly between the thighs. A two-inch opening rimmed with rubber is located in the chest region of the raincoat, to which can be attached the suction hose from a specially designed vacuum pump. This has been fitted with a valve that permits it not only to create, very rapidly, an intense vacuum by sucking air out of the raincoat enclosed area, but also alternately permits air to reenter the

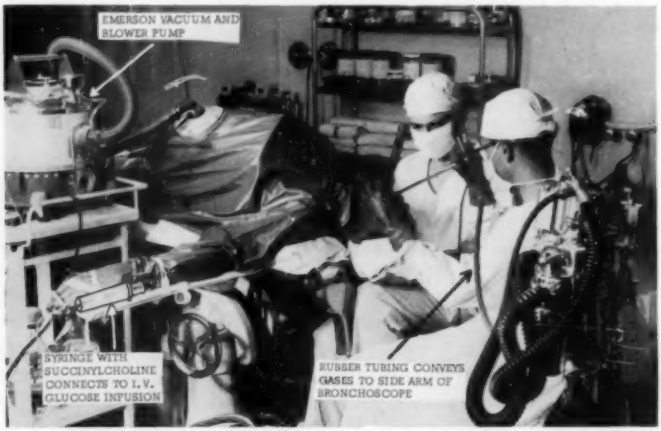

Fig. 1. Bronchoscopy. Patient is maintained in state of total paralysis by intermittent I.V. injections of succinylcholine. Light anesthesia is maintained by insufflating a mixture of N_2O+O_2+Cyclo through side arm of bronchoscope. Patient's head held by assistant. Respirator will maintain efficient respiratory exchange indefinitely. Leakage around patient's neck, wrist and thighs is minimal and of little significance, since vacuum causes raincoat to adhere to skin.

area, thus establishing a ventilation pattern which maintains not only tidal volume, but may even be used to hyperventilate the patient. The suction causes expansion of the chest wall which produces inhalation, while the alternate re-entrance of air back into the cage causes the chest to fall, producing exhalation. A patient lightly anestheteized and totally paralyzed with succinylcholine can thus be ventilated with complete efficiency.

There is no limitation to the length of time the operator

can work, and he can proceed in a leisurely and thorough manner, not being hurried by discomfort or fear for the condition of the patient. A laryngoscope with a Lewy laryngoscope holder (see Fig. 2) can be rested on the patient's chest wall, thus allowing operations on the larynx while the operator has two hands free for maneuvers. With succinyl-

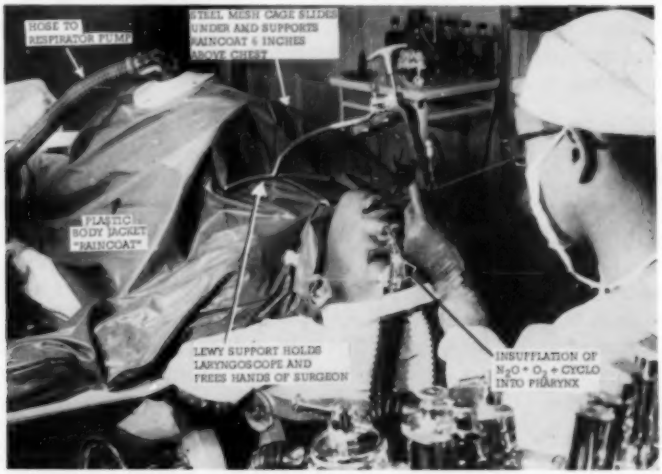

Fig. 2. Laryngeal Surgery. Light anesthesia is maintained by insufflating N_2O+O_2+Cyclo through a naso-pharyngeal tube, the tip of which lies at the uvula. Paralysis is maintained by intermittent injections of succinylcholine. A Lewy support holds laryngoscope, which frees surgeon's hands. Respirator breathes for patient by creating first a negative, then a release of pressure around chest wall.

choline the larynx is at rest in abduction, leaving the free margins of the vocal cords along with the subglottic region easily visible.

Esophagoscopies are facilitated and safer on a completely relaxed patient with better visibility and less chance of traumatic perforation.

Bayuk[5] was the first to describe the use of a polio chest shell for maintaining respiratory exchange in curarized patients subjected to peroral endoscopy; however, the chest

shell elevates and depresses only the anterior portion of the chest wall, and does not produce the large tidal exchange possible with the Emerson plastic "raincoat" cage.

BRONCHOSCOPY.

The patient first dons the plastic raincoat, or poncho, and after placing the steel mesh cage over the chest (under the raincoat) the neck and arm strings are tightened to prevent leakage of air in or out. An intravenous glucose infusion is first established so that a patent vein will always be available to maintain succinylcholine paralysis.

The patient is then lightly anesthetized by any favorite method; then rendered apneic by succinylcholine and the respirator turned on. The bronchoscope is then passed into the trachea while oxygen, with anesthetic gases are continuously administered through the side-arm of the instrument. It is advisable to pass the bronchoscope quickly, since the extreme relaxation may produce obstruction of the glottis because of a relaxed epiglottis. It is sometimes more efficient to pass the bronchoscope through a laryngoscope for this reason. Adequate exchange prevails throughout the state of paralysis. If the patient reflexly moves additional succinylcholine is injected.

LARYNGEAL SURGERY AND ESOPHAGOSCOPY.

In laryngeal surgery anesthetic gases must be administered by insufflation to maintain unconsciousness, and this is done by means of a nasopharyngeal tube, which does not enter the trachea.

For laryngoscopy and laryngeal surgery the vocal cords are continuously under direct observation since they are the surgical target, and patency of the airway is thus assured by direct vision.

In esophagoscopy, on the other hand, airway patency is not assured, and partial obstruction may exist even though the respirator is operating efficiently. In order to assure airway patency during esophagoscopy a stethoscope should be attached to the patient's neck at the larynx with adhesive,

so that ventilation can be continuously monitored. This is essential, since succinylcholine paralysis produces complete relaxation of the tongue, which often falls posteriorly to obstruct the airway even though the esophagoscope is in place. In order to prevent tracheal collapse from the instrument, an endotracheal tube may be inserted via the nose prior to introducing the esophagoscope, and the anesthetic gases delivered through this naso-endotracheal tube.

ANESTHESIA TECHNIQUE.

Patients may be induced with methitural (Neraval), hexobarbital (Evipal), or thiopental (Pentothal). We prefer methitural, since there is less postoperative inebriation. In addition, the return to consciousness occurs much more rapidly than with the other barbiturates.

The induction dose of the three drugs in the average adult is:

Methitural—10 cc. of a 4 per cent solution.

Hexobarbital—10 cc. of a 4 per cent solution.

Thiopental—8 cc. of a 4 per cent solution.

As soon as the methitural is injected a mask is placed on the patient's face, and a mixture of nitrous oxide, cyclopropane and oxygen is administered in a semi-closed system as follows:

Cyclopropane—700 cc. per minute (11.7 per cent).

Nitrous oxide—2000 cc. per minute (29.8 per cent).

Oxygen—4000 cc. per minute (58.5 per cent).

This mixture of gases will produce light, plane I anesthesia within three to five minutes, and no matter how long it is administered a level of anesthesia deeper than plane I will be impossible to achieve. We have used this exact mixture in over 30,000 patients during the past nine years for every type of surgery. Details of its administration have been described fully elsewhere.[6,7,8]

When plane I is established, 20 mg. of succinylcholine is

injected through the intravenous infusion needle. At the occurrence of respiratory arrest the Emerson respirator is turned on, and the breathing bag on the anesthesia machine is observed so that inflation and deflation occurs with each respiratory cycle. While still under succinylcholine paralysis the mask is removed from the face, and the bronchoscopist inserts the bronchoscope quickly. To the side-arm tube of the bronchoscope is attached a piece of wide bore rubber tubing, through which is insufflated exactly the same mixture of nitrous oxide, oxygen and cyclopropane. If the patient should reflexly move, or if added relaxation is requested, an additional cc. of succinylcholine (20 mg.) is injected intermittently whenever necessary. When the operation is over, pure oxygen is insufflated through the bronchoscope for about two minutes prior to removing it. When the patient's own respiratory efforts resume the Emerson respirator is turned off.

For laryngeal surgery the same mixture of gases is insufflated through a nasopharyngeal tube which is inserted through the nose, only to the uvula. In esophagoscopy a stethoscope should be taped to the neck in the region of the larynx so that respiratory exchange can be monitored, in the event an endotracheal tube is not used.

SUMMARY.

A new method of assuring adequate ventilatory exchange during bronchoscopy, esophagoscopy and laryngeal surgery under light general anesthesia, with full curare paralysis, has been described. An Emerson body jacket raincoat over a steel mesh cage is employed in conjunction with a special vacuum pump respirator to provide intermittently expansion and compression of the chest wall, which thereby assures efficient ventilation.

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INTERNATIONAL VOICE CONFERENCE.

The International Voice Conference, dealing with laryngeal research, physiology, and therapy, was held May 20-22, 1957, in Thorne Hall on the Chicago Campus of Northwestern University, under the joint chairmanship of Hans von Leden, M.D., and Paul Moore, Ph.D., with the co-operation of representatives from all universities in the Chicago area.

Delegates from twenty-five foreign countries and the United States represented the leading laryngeal research centers. Thirty-eight scientific papers covered all aspects of laryngeal research. The guest of honor, Dr. Eelco Huizinga, Professor of Otolaryngology at the University of Groningen in the Netherlands, addressed the formal closing ceremonies on the subject: "The Larynx and Voice—Animals and Man."

At the same meeting Mr. W. E. Gould, President of the William and Harriet Gould Foundation, announced an annual award for outstanding research in the field of laryngology. First recipient of this award was Dr. K. Faaborg-Andersen of Copenhagen, for his electro-myographic studies of internal laryngeal muscles in humans.

TRACHEOTOMY: ITS PRESENT DAY APPLICATION.

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and
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The evolution of tracheotomy during recent years has extended the collaboration of the otolaryngologist with all phases of medicine and surgery. The numerous conditions of secretory obstruction of the lower respiratory tree, which have been favorably influenced by tracheotomy, and the development of new surgical techniques related particularly to the progress of anesthesia, as well as the advent of antibiotics, have created a new criteria for elective or therapeutic tracheotomy.

An understanding of the pathologic physiology of the mechanism of upper and lower respiratory obstruction, and its influence on the health status of the individual, is the keynote in the *urgency* of adequate treatment in order to avoid asphyxia. Interference with an efficient tracheobronchial airway is frequently present as a secondary complication, and early recognition and treatment reduces the morbidity and mortality of the many clinical conditions.

The purpose of this paper is to analyze the physio-pathology of lower respiratory obstruction, the many conditions related, the effects and limitations of tracheotomy, and the management of tracheotomized patients and treatment of the complications.

PHYSIO-PATHOLOGY OF RESPIRATORY OBSTRUCTION.

The indications for tracheotomy are a corrollary to the physio-pathologic obstructive changes in the upper and lower airways (Scheme I).

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Nov. 15, 1957.

INDICATIONS FOR TRACHEOTOMY

A. FIXED OBSTRUCTION TO UPPER AIRWAY :

CONGENITAL ANOMALIES
TRAUMA
INFLAMMATION, ACUTE AND CHRONIC
NEOPLASMS
FOREIGN BODIES
PARALYSIS
EXTRINSIC LARYNGEAL DISEASE

B. SECRETORY OBSTRUCTION TO LOWER AIRWAY :

1. PRIMARY NEURO-MUSCULAR DISTURBANCES :
   POLIOMYELITIS
   TETANUS
   BOTULISM
   MYASTENIA GRAVIS
   BULBAR OR PERIPHERAL NEUROLOGICAL LESIONS.

2. SEVERE DEBILITATION AND COMA :
   SYSTEMIC DISEASES
   GENERALIZED INFECTIONS
   POISONING AND TOXIC STATES
   CEREBRO-VASCULAR ACCIDENTS
   SEVERE TRAUMA WITH BRAIN INJURIES
   NEUROSURGICAL PROCEDURES.

3. MECHANICAL FACTORS OF VENTILATION :
   THORACIC SURGERY
   MULTIPLE RIB FRACTURES
   PERSISTENT MEDIASTINAL EMPHYSEMA.

Scheme I.

The normal protective mechanisms of the tracheobronchial tree that prevent the aspiration and favor the expulsion of excessive secretions and foreign materials are: *1.* the reflex of deglutition; *2.* the ciliary activity; *3.* the peristaltic action, and *4.* the cough reflex. The alteration of any of these mechanisms, either singly or together, will result in an inefficiently functioning tracheobronchial airway with interference of adequate oxygenation.

Though the mechanisms of secretory obstruction are often associated in a variety of pathological states, three principal groups of conditions should be considered:

1. Those in which prevails a neuro-muscular disturbance, central or peripheral, as in poliomyelitis, tetanus, botulism, myasthenia gravis, and other bulbar or peripheral neurological lesions on an inflammatory, neoplastic, degenerative, vascular or traumatic basis.

2. Conditions of serious debilitation and coma in which prevails the depression of the central nervous system, as in systemic diseases, generalized infections, and poisonings.

3. Mechanical factors of the thoracic cage, as in postoperative and post-traumatic thoracic alterations.

In poliomyelitis, the respiratory disturbance may be central or peripheral in origin. Central involvement is on a viral basis, and the nuclei of the respiratory musculature are involved in their spinal and bulbar sections. The peripheral disturbance is mainly related to three factors: *1.* The mechanism of artificial respiration; *2.* The absence of the cough reflex; *3.* The altered deglutition.

The paralysis of respiratory musculature renders inspiration and the increase in intrathoracic pressure inadequate for an effective cough. If a patient in a respirator can cough effectively, there is not a complete paralysis of the thoracic and diaphragmatic musculature. If the chest is immobile and the patient must adjust to the rhythm of the respirator in which there is no respiratory arrest at the time of deglutition, the aspiration of abnormal material in the tracheo-bronchial tree is a common occurrence. Such a complication is definitely facilitated in the bulbar form with paralysis of the buccal, pharyngeal, esophageal and laryngeal musculature. In these cases only the tracheotomy enables one to bypass the pooling of material in the pharynx and prevent asphyxia.

In tetanus, there is a peripheral neuro-muscular dysfunction of respiratory musculature associated with lesions of the respiratory, deglutition and cough centers. Respiration be-

comes irregular with frequent periods of apnea; the mechanism of deglutition is altered with pharyngeal pooling of secretions, and the cough is very weak or absent. Laryngeal spasms and deep sedation are important factors in respiratory obstruction.

Botulism results in a weakness of the respiratory musculature, ineffectiveness of the cough mechanism and a disturbance of deglutition, which is related to blockage at the level of the motor end plates of nerves.

Myasthenia gravis is a disturbance of the myo-neural synapse with extreme weakness of musculature. A bulbar syndrome characterized by inadequacy of mastication, deglutition, phonation and respiration may be present. The cough reflex may be inadequate, with resultant accumulation of pulmonary secretions and pneumonia *ab ingestis* may develop.

In the patients seriously debilitated and comatose, and in postoperative and post-traumatic pulmonary complications, lower respiratory obstruction is due to an accumulation of secretions and abnormal material in the tracheo-bronchial tree associated with an inadequate expulsive mechanism.

Pharyngeal aspiration may occur under general anesthesia or following major procedures around the mouth, pharynx and neck, in which there is a disturbance of deglutition due both to neuro-muscular lesions and to the pain experienced. Similarly, after cerebral trauma or endocranial procedures, there may be an involvement of the IXth and Xth cranial nerves with paresis of pharyngeal musculature and absence of pharyngeal reflex. On the other hand, there is a profuse salivation, due to the stimulation of the salivatory nuclei and irritation produced by the Levine tube.

Excessive bronchial secretions may occur after surgical procedures as a response of the respiratory mucous glands to prolonged irritation of the endotracheal tube and anesthetic gas. Such hypersecretion is of importance in thoracic surgery, in which the broncho-pulmonary lesions determine accumulation of secretion and in which the resection of seg-

ments and the collapse of a lung cause limited pulmonary ventilation.

The suppression of the cough reflex seems to be the most important factor in secretory respiratory obstruction. It occurs under general anesthesia, and in comatose states due to systemic diseases, generalized infections and poisoning. In particular, eclampsia, barbiturate and morphine poisoning, uremia and serious burns have been reported. A comatose state with inadequacy of the cough mechanism may follow serious trauma, endocranial operations and cerebro-vascular accidents.

Mechanical factors may be responsible for the ineffectiveness of the cough mechanism, as in elderly patients with inadequate muscular strength, pulmonary emphysema and fibrosis. On the other hand, if the movements of the thoracic cage are a source of pain, the patient will automatically reduce the respiratory movements. In children, there may be a lack of cooperation on the part of the patient.

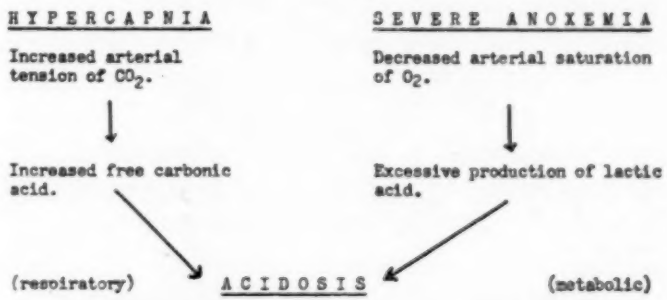
Special conditions are represented by altered mechanisms of the thoracic cage, such as multiple fractures of the ribs and thoracoplasty. In the flaccid thorax a part of the wall moves paradoxically in relation to normal expansion, and is often associated with limited movement of the chest, and with the pain may determine inadequacy of the cough reflex. Persistent mediastinal emphysema after a trans-sternal approach to the mediastinum may cause an unbalance in the thoracic negative pressure with consequent respiratory obstruction.

The accumulation of secretions and abnormal materials in the tracheo-bronchial tree produces two types of physiopathological effects: local—on the pulmonary structures, such as atelectasis and later atypical pneumonia and pulmonary abscess; systemic—biochemical changes leading to asphyxia.

The lower secretory obstruction determines an increase in the negative intrathoracic pressure with subsequent passive dilatation of the pulmonary capillaries followed by congestion, edema, hemorrhage and exudation. Similarly, anoxemia is often the cause of vasodilatation, increased capillary permeability and pulmonary edema. The load on the heart is

increased and decompensation may develop. If the bronchi or bronchioles are obstructed, air is reabsorbed in the distal segment, and atelectasis develops associated with compensatory emphysema of the surrounding segments. Secondary infection of the atelectatic area may result in atypical pneumonia, pulmonary abscess and bronchiectasis.

These considerations are important to show that secretory obstruction at the level of the tracheo-bronchial tree is not only a problem of anoxia to be solved with administration of



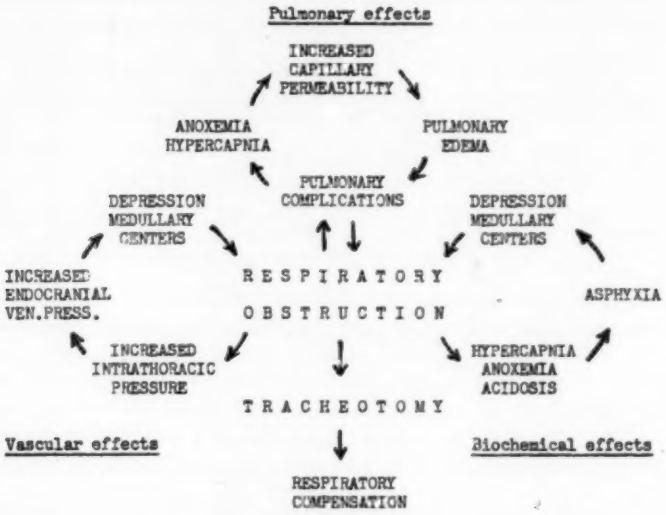
Scheme II.

oxygen, but also that progressive and irreversible pulmonary changes may develop, making any therapeutic attempt useless.

The systemic effects of respiratory obstruction are characterized, first by a decrease of pulmonary ventilatory capacity and then by a decrease of respiratory equivalent for oxygen. The ratio between the volume of inspired air and the volume of oxygen used in one minute decreases. Normally, hyperventilation could correct such a deficiency, but in respiratory obstruction muscular effort causes a greater use of oxygen and aggravates the existing condition.

The biochemical changes that follow are characterized by: *1.* Hypercapnia with increase of the arterial tension of carbon dioxide; *2.* Anoxemia with a decrease of arterial saturation of oxygen, and *3.* Acidosis as a consequence of hypercapnia and anoxemia. (Scheme II).

The asphyxia due to respiratory obstruction is a summation of the above three factors. It is due not only to anoxia, and cannot be avoided by the simple administration of oxygen. Hypercapnia and anoxemia cause cerebral vasodilatation that, associated with venous congestion (edema) due to the increased negative intrathoracic pressure, produces endocranial hypertension with depression of the medullary centers of respiration and cough.



Scheme III.

The several vicious cycles that correlate local and systemic, vascular and biochemical effects of respiratory obstruction are demonstrated in Scheme III. The tracheotomy with removal of the secretory obstruction may break these cycles and reestablish a new gaseous exchange in the pulmonary alveoli with absorption of oxygen and elimination of carbon dioxide.

An important moment in respiratory obstruction is represented by the so-called "crisis." Hypercapnia and acidosis, at the beginning, have a stimulating action on the medullary

centers and cause hyperventilation. By increasing the concentration of carbon dioxide there will be a period of agitation, intolerance and then a period of narcosis and depression. The critical point of inversion of the effect of carbon dioxide is lowered by the depression of the centers due to anoxemia, acidosis or administration of sedatives. The respiratory effort decreases, and respiration stops.

The administration of oxygen may help the centers to stand the inhibitory effort of carbon dioxide and metabolize excessive lactic acid; however, it will not modify hypercapnia and respiratory acidosis. In some cases the administration of oxygen may be fatal. When there is severe depression of the centers due to anoxia or excessive sedation, the only respiratory stimuli are represented by anoxemia, which acts on the aortic or carotid chemoceptors that become the peripheral respiratory centers. By eliminating anoxemia, apnea develops and artificial respiration becomes necessary until the centers reassume their excitability. Only the removal of respiratory obstruction will avoid fatalities.

RESPIRATORY OBSTRUCTION AND TRACHEOTOMY.

The prophylaxis of accumulation of secretion in the tracheobronchial tree and pulmonary atelectasis after major surgical procedures is of utmost importance. It consists of an adequate anesthesia with early return of the cough reflex, aspiration of the tracheo-bronchial tree, hyperventilation in the immediate post-anesthetic period, frequent change of position, early ambulation and prophylactic antibiotic therapy.

When a lower secretory obstruction has developed, the treatment may be conservative or surgical. The conservative treatment may consist of oxygen therapy, postural drainage and tracheobronchial aspiration through an endotracheal tube or bronchoscopy.

Though the conservative treatment may be quite useful in some cases, it usually requires an adequate set-up and well trained personnel. If this is not available it may be necessary to perform a tracheotomy in order to have a better control of the situation at all times.

Tracheotomy, from a general point of view, may be: *1.* Therapeutic, if there is respiratory obstruction that cannot be treated conservatively; and *2.* Elective, if the respiratory obstruction is not yet present, but is imminent, and may impose a penalty on the general and local conditions of the patient.

The tendency is to perform an elective tracheotomy in doubtful cases. According to our experience, it is much safer to perform a tracheotomy than to wait; it is much easier to keep the airway patent and free of secretion and, therefore, prevent further complications.

Tracheotomy is no longer a feared and dangerous procedure, providing the surgeon is aware of the surgical anatomy. Mortality due to tracheotomy is less than one per cent.

The effects of tracheotomy may be purely mechanical and can be explained on physical laws; or they may influence the mechanism of pulmonary ventilation and its physiologic regulation. These can be summarized as follows:

1. To bypass an obstruction of the upper airways due to edema, hemorrhage, neoplasia, foreign body and functional disturbance of the larynx.

2. To bypass secretions and abnormal material accumulated in the pharynx and avoid their aspiration in the tracheobronchial tree.

3. To enable a patient in a respirator to swallow independently from the uninterrupted cycle of aspiration and compression.

4. To facilitate aspiration of secretions from the tracheobronchial tree, even by non-trained personnel.

5. To facilitate the elimination of crusts and thick and viscid material by the introduction in the trachea of saline solution.

6. To avoid the formation of pulmonary atelectasis due to bronchial obstruction; in complete obstruction of a bronchus, the increased intrathoracic pressure during the cough mechanism will displace the mucus plug more peripherally, caus-

ing atelectasis; by reducing the power of the cough mechanism, the removal of the mucus plug is left to ciliary activity, peristaltic bronchial action and aspiration.

7. To reduce the dead space and allow a more useful utilization of the respiratory air, sometimes reduced to a value incompatible with normal ventilation.

8. To decrease the resistance offered to inspiratory and expiratory air in the nasal, pharyngeal and laryngeal cavities, thus making pulmonary function more efficient. As a corollary in thoracic lesions, there will be a reduction of pain in the respiratory movements and a decreased paradoxical excursion of the flaccid thoracic segments.

TECHNIQUE OF TRACHEOTOMY.

Three techniques may be used according to the indication for tracheotomy: emergency, ordinary, permanent.

The position of the patient with the neck hyperextended and the palpation of the landmarks of the neck before the procedure are of extreme importance. No anesthesia is necessary if the patient is comatose or unconscious. Local anesthesia is the most widely used; general anesthesia with endotracheal intubation is limited to selected cases. The insertion of a bronchoscope in children is very helpful. When possible, the administration of oxygen by mask or through an endotracheal tube or bronchoscope may be of considerable value.

In urgent cases a longitudinal incision in the midline extending from the upper border of the cricoid cartilage to the sternal notch is employed. Two Kochers are applied longitudinally at the border of the skin incisions for hemostasis. The anterior aspect of the trachea is looked for at the inferior border of the cricoid cartilage in the midline, which is the best topographical surgical landmark. Two more Kochers are inserted on the anterior aspect of the trachea in a paramedian position so as to clamp all the soft tissues overlying the trachea. A longitudinal incision is performed between the Kochers and the trachea is severed longitudinally at the level of the second and third tracheal rings. The cannula is

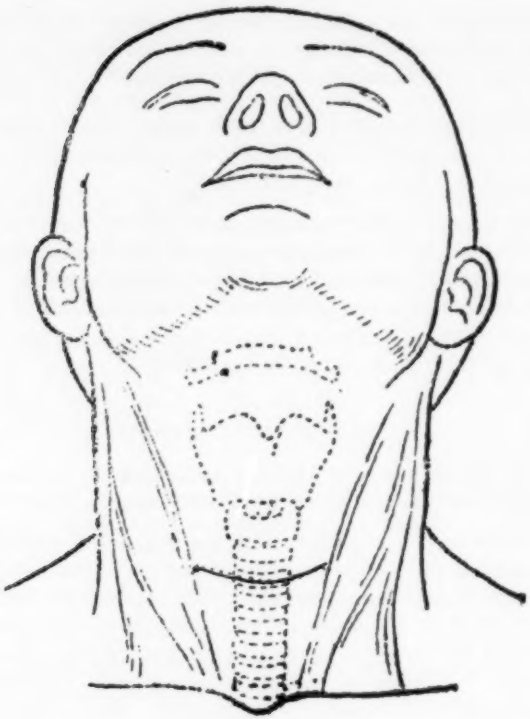


Diagram I.

Collar incision for tracheotomy and landmarks of the neck.

inserted, bleeders ligated and loosely placed skin sutures are used.

In an ordinary tracheotomy, when there is no acute respiratory obstruction, a "collar" incision has proved quite superior to a longitudinal one. With a collar incision it is actually much easier to identify the space between the strap muscles of the neck and avoid useless dissection into the muscles. If the patient is in a respirator or has a short, fat neck a collar incision is much easier. Finally, the cosmetic results are far superior; however, great care should be taken to avoid severing the longitudinal vessels of the neck with the skin incision.

A collar incision is performed at approximately two centimeters below the interior border of the cricoid cartilage, from the anterior border of one sterno-cleido-mastoid muscle to the other, according to diagram 1. The incision includes skin and subcutaneous tissue down to the anterior aspect of the sterno-hyoid muscles. The two skin flaps are elevated for approximately two centimeters.

A longitudinal dissection between the strap muscles of the neck follow and the sterno-hyoid and sterno-thyroid muscles are retracted laterally with rakes. The isthmus of the thyroid gland is identified at the inferior border of the cricoid cartilage, where it is clamped by two Kochers and severed longitudinally. The trachea is then incised longitudinally in the midline at the level of the second and third tracheal ring and a crescent piece of cartilage is removed on either side of the incision. The cannula is inserted.

If the patient is in a respirator it is important to perform a high incision, avoid dissection of muscles with controlled hemostasis, because the irritation of the collar of the respirator may cause subcutaneous and mediastinal emphysema and annoying hemorrhages.

If the tracheotomy is going to be permanent, a tracheostomy is indicated. By tracheostomy it is intended to suture the edges of the skin incision to a window performed on the anterior wall of the trachea. It may be done with a longitudinal or collar incision. The basic principle is to bring the skin to the trachea by elevation of the skin flaps. A tracheostomy reduces the danger of emphysema or infection, and decannulation or recannulation will not present a problem.

MANAGEMENT OF TRACHEOTOMY.

The postoperative management of tracheotomized patients is of utmost importance; without accurate care, fatal pulmonary complications may develop. The airway must be kept patent and free of secretions, crusts and abnormal material continuously. Management includes general care of the patient, care of the tracheostomy and proper humidification and oxygenation (Scheme IV).

The general care of the patient consists of bed rest for at least 24 hours, possibly with elevation of the head of the bed to facilitate breathing. The diet should be given as tolerated, orally or parenterally. Anodynes and sedatives are administered as needed, but avoiding diminution of the cough reflex to prevent the formation of mucus plugs.

The emotional aspects of tracheotomy are most certainly an integral part of the general care. Tracheotomized patients

MANAGEMENT OF TRACHEOTOMY

GENERAL CARE OF PATIENT

BED REST
DIET
SEDATIVES
ANODYNES
ANTIBIOTICS
EMOTIONAL ASPECTS

CARE OF TRACHEOSTOMY

EQUIPMENT AT BEDSIDE
ASPIRATION OF SECRETIONS
TRACHEOTOMY CANNULAS
DRESSINGS

HUMIDIFICATION AND OXYGENATION

Scheme IV.

are extremely apprehensive relative to the loss of their voices as well as the new mechanism of breathing through a tube in the neck. These patients need reassurance and should be instructed to place a finger over the opening of the tracheotomy tube whenever they desire to talk.

The care of a tracheostomy requires certain equipment at the bedside. An aspirator should be available with sterile saline and antiseptic solutions. A rubber catheter should be kept in the antiseptic solution when not in use. A Trousseau dilator and a sterile tracheotomy tube of the same size used for the initial tracheotomy should be at hand. The tracheo-bronchial tree should be aspirated frequently and

adequately; the inner tube should be removed and thoroughly cleaned with soap and water as needed, and the position of the cannula should be checked often for patency and presence of decubitus on the tracheal wall.

The dressing around the tube should be changed as needed. The outer cannula should not be removed until five days after the procedure unless a tracheostomy was performed. The hygiene of the skin around the tracheostomy is important to avoid secondary infection.

COMPLICATIONS OF TRACHEOTOMY

RESPIRATORY OBSTRUCTION
HEMORRHAGE
EXPULSION OF CANNULA
SUBCUTANEOUS EMPHYSEMA
LOCAL INFECTION
HYPERVENTILATION
MEDIASTINITIS

Scheme V.

The humidification may be accomplished with a humidifier, croupette, or steam room. When thick secretions or crusts are present, wetting agents should be administered (saline or aerosols). Oxygenation is indicated when hypoxia occurs on a nonobstructing basis.

The frequent complications of tracheotomy (Scheme V) are those of infection, bleeding, respiratory obstruction (particularly in children), encrustations, and emphysema. The majority of these conditions are treated primarily from a prophylactic aspect.

Cleanliness in the handling of the tubes (use of alcohol, boiling and soap and water) helps to eliminate areas of infection and subsequent crust formation. The cannula should be handled gently to avoid excessive bleeding, and the frequent aspiration and cleansing of the inner cannula will greatly reduce the incidence of encrustation and respiratory obstruction.

One of the most difficult problems to cope with is that of encrustation, which has an incidence of about 10 per cent, and usually becomes evident one to two weeks following the procedure. The control of pulmonary pathology, sinus disease and upper respiratory infection reduces the severity of such a complication. This can also be diminished with the use of proper humidification, proper nourishment, antibiotic therapy, the elimination of trauma, and the aid of wetting agents such as aerosols and saline flushings.

SUMMARY AND CONCLUSIONS.

Three factors are essential for the success of every surgical procedure but particularly for tracheotomy: proper timing, accurate surgical technique, and adequate postoperative care. It is quite obvious that failures after tracheotomy are mainly due to unjustified delay, urgency of the situation with an inadequate set-up and inefficient postoperative management. Certainly a tracheotomy cannot alter the prognosis of a patient in extremis, or when asphyxia and cardiovascular collapse are irreparable.

It is paramount that early tracheotomy be performed to avoid asphyxia, pulmonary complications and cerebral edema secondary to respiratory obstruction. This is accomplished by facilitating aspiration of secretions and maintenance of an adequate airway; however, one must bear in mind that there must be justification for such a procedure, since the physiology of the pulmonary tree becomes altered, the cough reflex is rendered relatively ineffective, and an additional surgical procedure in the presence of a serious pathologic state may precipitate a "crisis."

The importance of lower secretory respiratory obstruction and its effect on the body is stressed, with particular reference to problems of pulmonary decompensation and the situation of the insidious development of cerebral edema, often the irreversible state.

The management of tracheotomy, the related complications

and their treatment, as well as the technique of the various procedures is considered.

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PROGRAM OF THE SEVENTH INTERNATIONAL CONGRESS OF BRONCHOESOPHAGOLOGY.

Meeting Place—Kyoto University, Kyoto, Japan.

Friday, September 12th, 1958

8:00 P.M.-10:00 P.M.—Reception (Party) Shimomura House in Kyoto.

Saturday, September 13th

8:00 A.M.—Inaugural Session (in Kyoto University Hall).
9:00 A.M.-12:00 M.—First Scientific Session.
12:00 M.-2:00 P.M.—Luncheon (Reception. The Place not decided).
2:00 P.M.-6:00 P.M.—Sightseeing in Kyoto.
8:00 P.M.—Banquet, Miyako Hotel.

Sunday, September 14th

8:00 A.M.-11:30 A.M.—Second Scientific Session.
11:30 A.M.-12:00 M.—Closing Ceremony.
12:00 M.—Departure for sightseeing in Nara.
1:00 P.M.—Luncheon, Nara Hotel.
2:00 P.M.-6:00 P.M.—Sightseeing in Nara.
7:00 P.M.—Return to Kyoto.

INTRA-AURAL MUSCLE CONTRACTION IN MAN, EXAMINED BY MEASURING ACOUSTIC IMPEDANCE OF THE EAR.*

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INTRODUCTION.

Inasmuch as the intra-aural muscles appear to be of some importance to the function of the ear, it is desirable to understand their behavior in greater detail. The contraction takes place bilaterally when one ear is stimulated by a strong tone, with a minute, but clearly measurable, delay in relation to the stimulating tone. The latent period before the reaction of the muscles is significant, in that the muscles must be regarded as inactive during this latency. The majority of the knowledge we have concerning the function of the intra-aural muscles has been obtained from animal experiments, as it has been difficult to study the action of the muscles in man. Usually the stimulus has been applied to one ear, while the other ear has been used for measuring the muscle contraction.

A report will be made here of an investigation of the contraction of the intra-aural muscles in man, carried out by using measurement of the acoustic impedance in the external auditory canal. Both the latent period of a contraction, shown as a change in impedance, and the degree of the impedance change as a function of the intensity of the stimulus will be studied.

EARLIER INVESTIGATIONS.

The function of the intra-aural muscles has been studied by many writers. The majority reach the conclusion that a muscle contraction causes a decrease in transmission, espe-

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Editor's Note: This manuscript received in the Laryngoscope Office and accepted for publication April 10, 1957.

cially with lower frequencies. Hallpike and Smith observed that a muscle contraction effects a decrease of the cochlear potential in cats. Wiggers (1937) studied guinea pigs, in which spontaneous contractions of the intra-aural muscles occur under light urethan anesthesia, and found a drop in the cochlear potential at lower frequencies when the muscles contracted. At 400 c/s a decrease of 20 db was observed; at 1100 c/s there was no change, and at 1500 c/s an increase in the transmission took place when the intra-aural muscles contracted. Above 2000 c/s there was no change. Galombos (1942) investigated bats and found activity of the intra-aural muscles at frequencies up to 55 kc/s. Death or anesthesia increased the cochlear potential for a strong signal, and this was taken by the writer to indicate that the intra-aural muscles affect a reduction in the transmission, and that the cochlear potential, therefore, increased when the muscles relaxed because of death or anesthesia. Wever and Vernon (1954) studied the cat, measuring the cochlear potential and finding that the reduction in transmission was very slight above 500 c/s but increased below that frequency. The writers state that the stimulating tone was only about 2 db above the contraction threshold. They observed, moreover, that the drop in transmission increased when the intensity of the stimulus was increased. When the stimulus was 20 db above the contraction threshold, a reduction of about 20 db was observed at 300 c/s. The stimulus frequency was 1000 c/s. With only tensor tympani active (with the stapedius tendon severed) there was practically no reduction in the transmission.

Metz observed that a contraction of the intra-aural muscles effects a change in the impedance of the ear. He found that the transmission reduction can be measured as a change in impedance in the external auditory canal, principally as a change in absorption. The angle of the reflection co-efficient remained almost constant during a muscle contraction. Using the same method, Jepsen studied the threshold value for a muscle contraction in relation to the auditory threshold. In order to determine which muscle caused the change in absorption, he examined seven patients with facial nerve paralysis, but was unable to demonstrate any change in im-

pedance accompanying stimulation of the contralateral ear. On the other hand he observed a normal impedance change in two patients with trigeminal paralysis. This he accepted as proof that the cause of a change in sound absorption is a contraction of the stapedius muscle and not of the tensor tympani. Jepsen further demonstrated that the threshold for muscle contraction observed with help of impedance measurement agrees closely with that obtained visually by observance of the stapedius tendon through a perforation in the tympanic membrane.

The various earlier studies of the latent period for a contraction of the intra-aural muscles were carried out, using a variety of methods which are reflected in the results. Kobrak (1930) studied rabbits and used flute tones as stimuli. As indicators for muscle contraction he employed myographs attached to the tendons of the muscles. He obtained a latency of between about 30 and 150 msec for the contraction of the stapedius, and between about 75 and 150 msec. for the tensor tympanic. Lorente de No (1935) studied the contraction of the tensor tympanic in rabbits under ether anesthesia. He used, as did Kobrak, myographs as indicators, but employed a better-defined stimulus in the form of a tone produced by a loudspeaker. As a result he obtained a latency of approximately 20 msec when the stimulus was about 15 db above the contraction threshold. When the stimulus was more than 35 db above the controction threshold, he observed a latent period of from 12 to 13 msec.

Tsukamoto (1934) examined rabbits under light urethan anesthesia. He used change in pressure in the labyrinthine fluid as an indicator of the contraction of the intra-aural muscles, recorded with the help of a capillary tube. He used a flute tone as stimulus. With this method he found a mean latency of 370 msec.

Perlman and Case (1939) registered action potentials from the tendon of the stapedius muscle in man in a contraction of the stapedius caused by stimulation with a tone at 1000 c/s applied to the contralateral ear. The intensity of this tone is described by the writers as "loud". Their results obtained on a number of experimental subjects gave a mean latency of

10.5 msec. Bornschein and Krejci studied rabbits, registering action potentials from the tensor tympanic muscle. The experimental animals were under local anesthesia. A click was used as stimulus. The latent period was found to be 12 to 13 msec.

Lorente de No and Bornschein and Krejci observed that the contraction of the intra-aural muscles in rabbits exhibited rhythmic changes (waves) in muscular tension and action potential, respectively. The frequency of these changes was

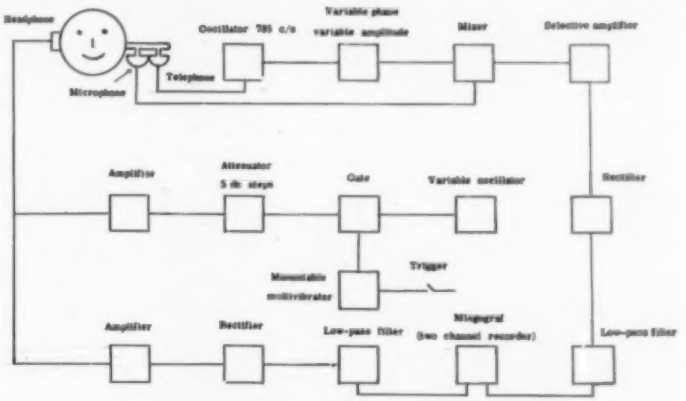

Fig. 1. Block-scheme of complete equipment.

15 to 20 c/s according to Lorente de No, and 15 c/s according to Bornschein and Krejci.

Metz (1951) investigated the latency of contraction in the intra-aural muscles in man by recording changes in the acoustic impedance of the ear, measured by means of a so-called Schuster bridge. He observed that the latent period was dependent upon the intensity of the stimulus. A stimulus 80 db above the threshold of hearing gave a latency of 150 msec, 90 db above gave 50 msec, and 100 db above gave

In order to find a closer relation between the latent period a latency of 40 msec.

and the intensity of the stimulus, as well as the variation in the results from person to person, the present investigation, using impedance measurement, was started. The reproducibility of the measurements was also investigated.

INSTRUMENTATION.

The equipment used was designed to deliver a stimulating tone of known frequency and intensity and to indicate a

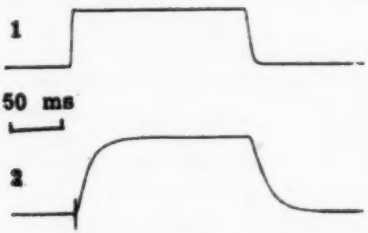

Fig. 2-a. Pulse response of the selective amplifier. The upper curve (1) represents a tone-pulse (785c/s) applied to the input of the amplifier, and the lower curve (2) represents the output from the amplifier. Both signals are rectified and low-pass filtered with 100 c/s cut-off frequency.

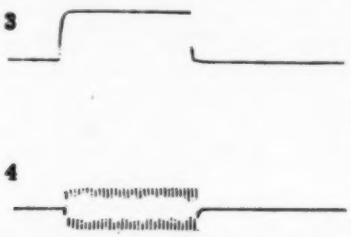

Fig. 2-b. The wave form of the stimulus pulse; (3) is stimulus 2500 c/s rectified and low-pass filtered with 700 c/s cutoff frequency; (4) is stimulus 250 c/s direct recorded.

muscle contraction. A muscle contraction was shown by recording changes in the acoustic impedance of the ear. The acoustic impedance was measured at a frequency of 785 c/s.

a. Fig. 1 shows a block-scheme of the equipment used. Stimuli are produced by means of an earphone of Permoflux type, PDR 10. The signal to the earphone is delivered by an oscillator with variable frequency which feeds the earphone via a gate circuit and an attenuator. A signal from a monostable multivibrator opens the gate in a manner that prevents the occurence of a click. The curve form of the tone pulse produced by the gate circuit is shown in Fig. 2.

The impedance change is indicated by means of a special measuring device consisting of a telephone and a microphone, both connected to a tube 4 mm in diameter which is inserted into the auricular canal. A signal at 785 c/s is fed to the telephone. The output voltage of the microphone represents the acoustic impedance in the ear measured in the external auditory canal. The output voltage is compared with a variable voltage having the same frequency. When this voltage has the same amplitude as the voltage arriving from the microphone, and opposite phase, the reading is 0. When the acoustic impedance changes, the voltage from the microphone also changes, and the resulting reading

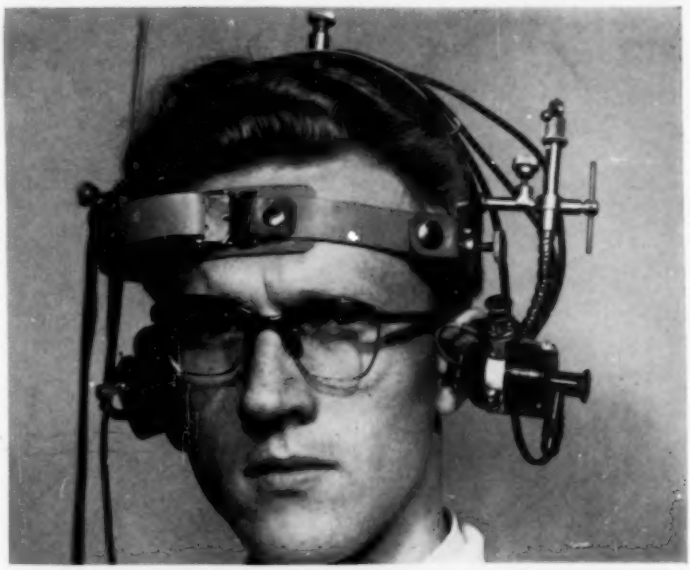

Fig. 3. The arrangement of headphone and impedance measuring unit in position on experimental subject.

will no longer be 0. This voltage corresponds to a change in impedance which is the result of a muscle contraction. The signal is amplified, rectified and filtered before it is fed into one channel of the recorder. In addition the signal to the earphone is amplified in the same manner as the signal that indicates a muscle contraction, and is thereafter fed to the other channel of the recorder. The latency is then evident as the time difference between these two signals. The prerequisite for this is that the apparatus be corrected for possible variations in the location of the loops in the recorder, and for delay in the selective amplifier amplifying the signal indicating a muscle contraction. The delay in the two low-pass filters used to filter the rectified signal has no significance, as the filters are the same in the two channels. Fig. 2 shows the curve form of an interrupted signal at 785 c/s, before and after amplification in the selective amplifier, recorded after rectification and filtration with

100 c/s low-pass filters in both channels (1) and (2). The curve form of a stimulus impulse is shown concurrently (3). The pulse shown is a rectified signal at 2500 c/s filtered only by the low-pass filtering action of the recorder. This means that the signal is distorted very little by the filters. Below this a stimulus signal at 250 c/s is reproduced directly (4). The recorder used (Mingograf) is a two-channel writing recorder, in which a fine ink jet traces a curve on ordinary adding machine paper. The frequency range of the recorder runs from direct current to 700 c/s. The paper feed rate used was 20 cm/sec in latency measurements and 5 cm/sec in registration of waves in the muscle contraction. The impedance measurement apparatus gives a sound pressure of about 75 db re 0.0002 dyne/cm². This is measured with subjective comparison.

b. The same equipment was used in measurement of absorption as a function of the stimulus intensity, except that the output of the microphone was measured directly in amplitude relation and phase relation to the voltage applied to the telephone. The voltage (E) from the microphone is in the following relation to the acoustic impedance Z_m:

$$Zm = k \cdot E \; ; \qquad Zm = \frac{Z \cdot Zi}{Z + Zi} \; ; \qquad Z = \frac{Zm \cdot Zi}{Zi - Zm} \; ;$$

and by using admittances:

$$Ym = \frac{1}{Zm} = \frac{1}{kE} \; ;$$

$$Y = Ym - Yi \; ; \qquad \left(Yi = \frac{1}{Zi} \text{ and } Y = \frac{1}{Z} \right) ;$$

The energy absorption a and the angle of reflection coeffecient R are determined from:

$$\sqrt{1-a} \cdot e^{j\varphi R} = \frac{\frac{1}{Y\varrho c} - 1}{\frac{1}{Y\varrho c} + 1} = -\frac{Y\varrho c - 1}{Y\varrho c + 1}$$

or

$$\sqrt{1-a} \cdot e^{j\varphi R} = \frac{\frac{Z}{\varrho c} - 1}{\frac{Z}{\varrho c} + 1} \; ;$$

$$a = 1 - \left| \frac{Y\varrho c - 1}{Y\varrho c + 1} \right|^2$$

where Z is the impedance of the ear and Z_i is the internal impedance of the microphone-telephone unit. The impedance of the ear is measured in the plane of the microphone. The absorption is most easily determined with the help of a bipolar chart, in which both impedance and admittance may be used. The use of admittances is advantageous because when admittances rather than impedances are used, the only effect of the internal impedance of the microphone-telephone unit is to shift the axes. In order to obtain the impedance of the ear in the plane of the tympanic membrane, it is necessary only to subtract the number of degrees corresponding to the length of the tube from the microphone to the membrane. The absorption in this bit of tube is so slight that it may be disregarded.

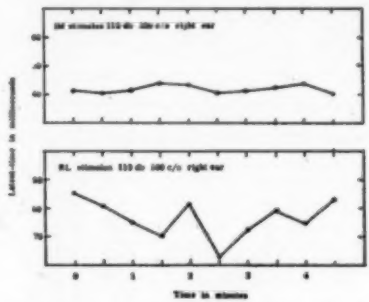

Fig. 4. The variation in latent time for a series of 10 measurements made, one every 30 seconds.

MEASUREMENT OF THE LATENT PERIOD.

Eight persons, ranging in age from 18 to 32 years, and all with normal hearing, were examined. The experimental subject was seated in a chair in a sound-insulated room. The stimulus was applied as a rule, to the right ear, and the impedance measuring unit was connected to the left ear. Fig. 3 shows the arrangement. The tube from the impedance measuring device was introduced into the ear. Vaseline was used to insure an airtight connection between the tube and the auricular canal. In measurements of the latent period a stimulus about 125 msec in duration was applied every 30 seconds. At least ten measurements were made at each intensity. It has been demonstrated that the latent period is not constant. In a series such as the one studied here, wide variations may be obtained (see Fig. 4). It was for this reason that several measurements were made at each in-

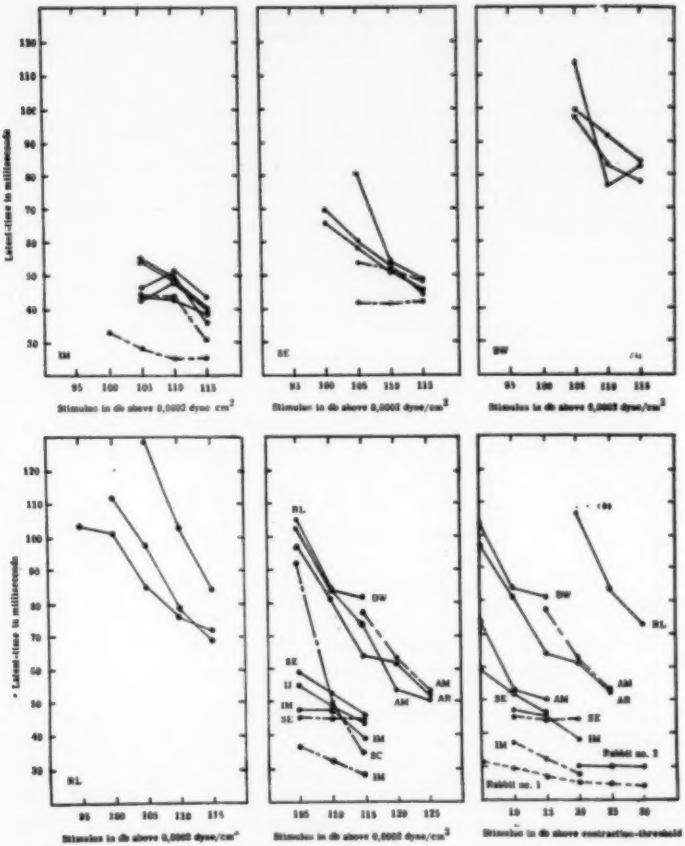

Fig. 5. The relationship between latent-time and stimulus intensity for different persons. Stimulus 1500 c/s, broken line. Stimulus 500 c/s solid line.

tensity rather than only one for each stimulus intensity. The latency has been defined as the mean value from a series of at least ten measurements. Immediately after, the next series of measurements was started, with the lowest intensity first, succeeded by increasingly higher intensities. This procedure was followed to avoid adaptation. The stimulus frequencies

used were 500 and 1500 c/s. The test tone used for impedance measurement was not interrupted between measurements.

Fig. 5 shows the relation between the intensity of the stimulus and the latent period and the reproducibility of such measurements. The different curves were recorded at intervals of several days. It is evident that there is great variation from person to person. The latency decreases as the stimulus increases at the same time that the reproductivity

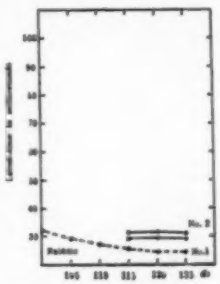

Fig. 6. The relationship between latent-time and stimulus intensity for two rabbits (there is no reference here between the cited intensity and 0.0002 dyne/cm^2).

of the measurements from day to day has a tendency to improve. The maximum and minimum latent time obtained was 130 and 25 msec. It appears that 1500 c/s stimuli gave a shorter latent period than 500 c/s. The intensity shown is re 0.0002 dyne/cm^2, measured in a 6 cc coupler, with the exception of the lower curve on the right (see Fig. 5), where an attempt has been made to give the intensity relative to the threshold of muscle contraction. This is rather unreliable, however, as the threshold value does not seem to be well defined, i.e., the threshold is much higher before a contraction than shortly after. The threshold discussed here was measured after a contraction caused by a tone somewhat higher than the threshold value.

In measurements on rabbits the same technique was used as that employed in the measurements on human subjects,

except that a tube was mounted on the telephone to carry the sound down into the rabbit's ear. Moreover, 2000 c/s was used as stimulus frequency (see Fig. 6). The tube on the impedance measuring unit was also longer. The rabbits were placed on a table and held in a cloth bag. An assistant held the telephone and impedance measuring unit in position. The rabbits were not anesthetized. The stated intensity of the stimulus was not re 0.0002 dyne/cm² in the measurements on rabbits because of the tube mounted on the telephone.

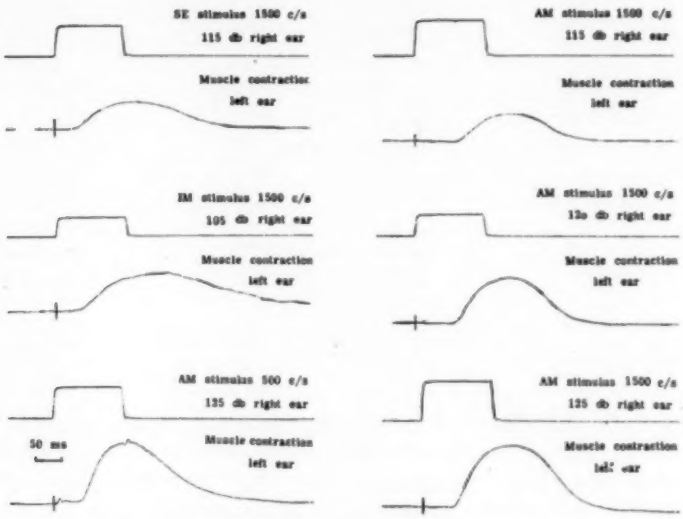

Fig. 7. Examples of recorded muscle contractions.

Fig. 7 shows examples of the muscle contractions produced by the different intensities and in different persons. The upper curve shows the stimulus and the lower curve shows the rectified signal that results from a muscle contraction. The deflection shown need not be in any definite relation to the degree of muscle contraction. This is because the registered signal occurs as the difference between the adjustable voltage and the output voltage of the microphone. There is no simple relation between the output voltage of the micro-

phone and the impedance or absorption of the ear. All that can be said is that when the microphone voltage changes it is due to a change in the impedance of the ear, and that may be due to a muscle contraction. It is not entirely impossible that a muscle contraction may take place without a change in impedance. Whether or not such a muscle contraction without a change in the impedance may be of any significance for sound transmission in the ear is uncertain.

Fig. 8. Examples of recorded muscle contractions with waves in the beginning of the contraction.

Lorente de No has demonstrated in experiments on rabbits that waves occur with constant frequency in the muscle tension when the tensor tympanic contracts. A similar phenomenon was observed in three of the experimental subjects in this study, and was most pronounced in SE. The waves were not apparent in stimulation with 1500 c/s, but were a constant phenomenon with stimuli at 500 c/s in these three persons. Fig. 8 shows examples of such waves. The frequency was relatively constant and was about 7 c/s. (Lorente de No ob-

served 15 and 20 c/s in rabbits.) The measurements in this case were carried out according to the same principles as the latent period measurements. The mean values of the ten measurements were:

SE stimulus—110 db 500 c/s right ear: 6.4 c/s
SE stimulus—110 db 500 c/s left ear: 6.6 c/s
SE stimulus—105 db 500 c/s left ear: 6.9 c/s
AM stimulus—120 db 500 c/s right ear: 7.4 c/s
AM stimulus—120 db 500 c/s left ear: 7.5 c/s
IM stimulus—110 db 500 c/s right ear: 6.8 c/s

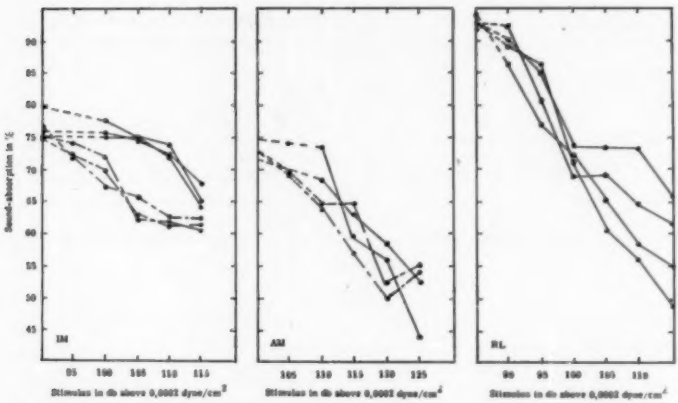

Fig. 9. The relationship[3] between stimulus intensity and sound absorption in the ear. The broken lines are for stimulus 1500 c/s and the solid lines are for stimulus 500 c/s. The points on the Y-axis, which are connected with dotted lines to the curves indicate absorption without stimulus.

Measurements on different days show that the reproductivity was extremely good.

The experimental subjects remarked spontaneously that the stimulus tone was not constant in amplitude at the beginning of the stimulation when 500 c/s was used. They heard a distinct change in amplitude on the occasions when waves were registered in the impedance change. The persons who did not exhibit these waves could not hear variations in the amplitude, not even when they had been instructed to listen for them.

Fig. 9 shows the sound absorption as a function of the intensity of the stimulus for three different persons, measured in the external auditory canal with a measuring frequency of 785 c/s. The different curves come from measurements on different days. The first is the lowest stimulus intensity used, followed by the next lowest and so on. The impedance changes in a muscle contraction appear to be a change in absorption without a change in the angle of the reflection co-efficient. The measurements show that a muscle contraction causes a decrease in the sound absorption, which takes place gradually as the stimulus increases.

SUMMARY.

The latent time for the contraction of the intra-aural muscles is measured in eight persons with normal hearing. The muscle contraction is demonstrated with impedance measurement. The latent time is defined as the mean of at least ten successive measurements at 30-second intervals. A wide variation in the results was obtained with respect to measurements, both on different persons and on the same person, with intervals of several days between measurements. For comparison, similar measurements were carried out on two rabbits. A phenomenon worthy of note was the observance of waves in the muscle contraction in three of the experimental subjects during stimulation at 500 c/s. The relation between the stimulus intensity and the sound absorption is investigated in three experimental subjects. These studies demonstrate that the muscle contraction causes a decrease in the sound absorption without a change occurring in the angle of the reflection co-efficient and that the decrease in absorption occurs gradually as the intensity of the stimulus increases.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

The American Board of Otolaryngology will conduct only one examination in 1958, and this will be October 6-9, 1958, in Chicago, Illinois, at the Palmer House.

For further information address Dr. Dean M. Lierle, Secy-Treas., University Hospital, Iowa City, Iowa.

A CASE OF UNUSUAL CONGENITAL FISTULA OF THE AURICLE.

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The detailed and informative article (Skokan) on the entire subject of congenital auricular fistulae in THE LARYNGOSCOPE of September, 1957, brings to my mind an unusual case I recently treated.

My patient, a female, aged 31, had a history dating back many years. She had several operations: some mere incisions with drainage of recurrent infected cysts; others, more determined attempts to irradicate her annoying and painful complaint.

She was not quite sure whether her trouble first started in the concha or post auricular region. When I first saw her she had a sinus that extended from the post auricular region to the junction of the concha and the floor of the external auditory canal. She informed me that it discharged thick pus continually, and was liable to recurrent attacks of acute inflammation with considerable pain, discomfort, etc. Gentle pressure with the finger-tip over the concha caused pus to present both anteriorly and posteriorly. This also caused acute pain, and the area where the sinus opened in the concha was very tender indeed. This, to my mind, indicated involvement of the perichondrium and cartilage of the concha; furthermore, the conchal sinus was surrounded by an area of necrotic looking skin about 1 cm. in diameter, and the post auricular area was also quite unhealthy looking and scarred from repeated surgery.

The problem was to cure the disease and preserve the appearance of the auricle as much as possible. I thought of injecting the sinus with radio-opaque oil, but saw little point in it.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication October 23, 1957.

After considerable thought it was decided to attempt a radical removal of the diseased tissue with subsequent skin grafting of the defects. A plan was devised to preserve the appearance of the auricle at operation.

Under general anesthesia a more or less circular incision was made surrounding the diseased conchal skin. The lateral part of this incision was carried laterally through the incisura intertragica, then caudally and posteriorly, and finally in a circular manner to surround the diseased post auricular region. This area was about 2 cm. in diameter. I might add that before the incision was made the posterior sinus was injected with Methylene Blue and the dye presented in the conchal sinus. The incision was deepened to expose the entire sinus tract. The supero-medial part of the sinus was surrounded by necrotic cartilage, and the circular incision was deepened to excise this cartilage. The entire tract was then excised with the attached upper and lower diseased skin and cartilage. The exact location of the postero-inferior end of the sinus was medial to the lobule over the lower part of the lateral surface of the mastoid process. The dye was a very useful guide to the depth and location of the sinus. The base of the wound was then curetted thoroughly.

The area was repaired as follows:

A split-thickness skin graft was cut from the medial aspect of the left thigh and used to repair the conchal and post auricular defect. The incision through the incisura intertragica and the attachment of the lobule to the skin of the face was then sutured. The conchal graft was made to fill its uneven bed with one-half inch vaseline gauze. The posterior area was dressed with non-sticking "Telfa". The entire area was left untouched for two weeks, and removal of the dressings showed that the grafts had taken very well indeed.

I have kept the patient under observation for several months; she has an execllent cosmetic result with no recurrence of any infection, and the grafts have survived exceptionally well. Her auricle looks normal and its configuration was not changed in any way by the operation. This is, of course, important, especially in a female patient.

THE ABSORPTION OF TOPICALLY APPLIED TETRACAINE AND COCAINE.*

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and
DONOVAN CAMPBELL, M.D.,
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LOCAL ANESTHETICS.

Local anesthetics rank high in the list of drugs which cause unnecessary deaths. Those who consistently use local anesthetics are familiar with their hazards; occasional users are often less versed in their pharmacology, toxicology and technique of use and usually experience the majority of difficulties.[1] The protocols of mishaps indicate that reactions and fatalities are most frequent after topical application. The otolaryngologists, in our experience, presumably because they use these drugs continuously and have respect for their potency, appear to encounter fewer difficulties.

The problem of reactions following the topical use of local anesthetics was of such concern that, in 1924, a commission was delegated by the American Medical Association to review the status of the then available drugs and make recommendations concerning dosage and technique of usage.[2] Although the situation has improved slowly since submission of their report, preventable deaths are still frequent. Seevers[3] has commented that more people die from local anesthetics than from any other group of drugs. Perhaps the problem is ignored, because the deaths occur sporadically, in many areas in the hands of many users.

Conflicting statements appear in the medical literature concerning absorption of local anesthetic drugs after topical application. Nearly everyone assumes that they are rapidly absorbed into the blood stream and that the high blood level

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Editor's Note: This manuscript received in the Laryngoscope Office and accepted for publication Nov. 4, 1957.

is the direct cause. Still, blood level studies to substantiate this assumption are not available. Eggleston and Hatcher[4] indicated the role of rapid absorption in causing reactions but reported no blood levels. Seevers and his co-workers,[5] studying the metabolism of cocaine, injected the drug subcutaneously, intravenously and into the stomach, but did not study absorption from the pharynx, trachea, bronchi and other mucous surfaces where it is used most often clinically.

We felt that blood level studies might provide a clue to the cause of the high reaction rate and undertook such a study. For obvious reasons, the study was done in the laboratory on dogs. Blood levels were determined after the application of tetracaine and cocaine, the two most serviceable and widely used drugs, to the mucous surfaces of the pharynx, trachea and bronchi. These blood levels were compared with those obtained after rapid intravenous injection, subcutaneous infiltration and slow intravenous infusion. The rapid intravenous injection of 6 mgm. per kilogram of tetracaine gave levels averaging 100 mgm. per ml. within one to two minutes. The same quantity spread over the pyriform fossae or instilled into the trachea resulted in peak levels of 30 mgm. per ml. The peak was attained within four to six minutes. The peak blood level was not so high, nor was it attained as abruptly, as that noted after rapid intravenous injection, but was far greater as compared to blood levels resulting from infiltration. There is some difference between the curves obtained after the pharyngeal and intratracheal instillations; however, it is not remarkable, and the peak levels of both curves far exceed those after subcutaneous injection or slow infusion. Epinephrine added to 2 per cent tetracaine did not retard absorption significantly. The blood levels did not differ remarkably from those obtained without the use of epinephrine.

In one set of experiments the total dose was doubled and applied in fractions at equal intervals of one minute over an eight to ten minute period. The blood level gradually attained a peak of 20 mgm. per ml. after ten minutes. The application of the drug in fractions resulted, as one would expect, in a gradual rise in blood levels. The blood levels were consider-

ably lower than those obtained after a single application. Identical blood levels were obtained if the total weight applied to a given area remained constant, even though the concentration was varied from 2 to 4 per cent. This is extremely important, because it indicates that the blood level is more dependent upon total dosage than concentration. The blood level was not detectable when 6 mgm. per kilogram of tetracaine in the form of a 0.1 per cent solution was infiltrated subcutaneously over an area of the abdomen 3 x 4 cm. The slow intravenous infusion of 6 mgm. per kilogram (.001 per cent solution) over a 20 minute period, likewise, caused no detectable quantity in the blood after the injection was complete.

When the free base was precipitated and applied to the mucous membranes, the blood level curves were identical to those obtained when comparable weights of the base in aqueous solutions of the hydrochloride were used. Absorption of the hydrochloride, incorporated in a water soluble cream base, applied to mucous membranes was prompt and rapid and occurred with ease. These data indicate that the topical application of a given dose of a local anesthetic results in blood levels similar to those obtained after rapid intravenous injection. The epithelial surfaces of the pharynx, trachea, alveoli and posterior urethra provide excellent absorbing surfaces. Drugs are readily absorbed from the pulmonary alveoli because the osmotic pressure relationships in the pulmonary vascular bed are designed to prevent collection of fluid in the sacs. Volunteers inhaling nebulized 4 per cent cocaine hydrochloride immediately experienced the stimulation characteristic of this drug and felt no appreciable numbness in the upper respiratory passages.

Similar studies were carried out using cocaine. The data closely follow the pattern of absorption obtained with tetracaine. The statement is heard that 10 per cent cocaine is less toxic than more dilute solutions, such as the 4 or 5 per cent, because the vasoconstrictor effect is more intense with the more concentrated solution and absorption is retarded. In order to determine whether or not this reasoning is correct, benzocaine crystals were incorporated in a 10 per cent

and a 4 per cent aqueous solution of cocaine and applied to the pyriform fossae. Should cocaine produce a vasoconstriction it should retard the absorption of benzocaine. Blood levels in each case were identical to those obtained when benzocaine was suspended in distilled water. When benzocaine crystals suspended in water were applied to the pharynx after first spraying with .05 norepinephrine to the point of blanching, blood levels averaging 80 mgm. per ml. were obtained after five to nine minute intervals with a gradual decline to the zero level after 20 minutes. Although these blood levels are somewhat lower than those of the control, and the peak is attained after a longer time interval, the difference is not so great that it is remarkable.

It appears that neither the vasoconstrictor effect of norepinephrine or of cocaine is sufficient to suppress appreciably the absorption of benzocaine from the mucous membranes. After the application of 100 mgm. of cocaine as a 4 per cent solution to the pyriform fossae the blood level rose to 8-12 mgm. per ml. within 4 to 6 minutes and gradually receded to undetectable levels within a period of 15 minutes. The same weight of cocaine as a 10 per cent aqueous solution applied to the pyriform fossae gave curves nearly identical in contour to those of 4 per cent cocaine. The peak was attained within six to eight minutes, and the maximum value was 10 mgm. With 4 per cent the maximum rise was within four to six minutes and rose to 8 to 12 mgm.; thus, even though some cocaine causes vasoconstriction this effect is insufficient to retard absorption significantly. When cocaine was combined with epinephrine nearly identical absorption curves were obtained. These were not remarkably different from those obtained with 4 per cent alone.

Dibucaine and tetracaine are the most potent and most toxic of the local anesthetics which possess suitable topical actions. Procaine, the least toxic local anesthetic drug in current use, is devoid of topical action. From the standpoint of effective topical anesthesia, one must concede that tetracaine is the most serviceable; however, one wonders whether he is justified in using it because of its toxicity. The term *potency* applied to a topical anesthetic is not easily defined.

Tetracaine is ten times more potent than procaine on the milligram for milligram basis; thus one mgm. of tetracaine should produce the same degree of blockade as 10 mgm. of procaine. On theoretical grounds, one would use 1/10 as much. Ordinarily one gram of procaine is the maximum quantity recommended for infiltration in an adult. On such a basis, it would follow that 100 mgm. of tetracaine would be the maximum used for infiltration. In topical anesthesia, however, this does not apply necessarily because the absorption after topical application to the mucous membranes is far more rapid than after subcutaneous injection. The total quantity, therefore, should be much less than that suggested for infiltration; how much less is difficult to say.

Carabelli has attained adequate anesthesia using a 0.25 per cent solution of tetracaine. He suggests 20 mgm. as the limit to be applied in fractional doses. Rubin and Culley[6] suggest 50 mgm. as the limit to be applied in fractions over a period of 15 or 20 minutes. Weisel and Tarrow[7] using quantities not exceeding 40 mgm. in 1000 endoscopy procedures noted 19 reactions. Seven of these were convulsive; the remainder were classified as mild circulatory. In the series of fatalities reported by the writers,[1] doses as high as 180 to 200 mgm. had been used in some cases. Obviously, these doses were excessive and presumably accounted for the difficulties. Cocaine is approximately five times more toxic than procaine; thus, on theoretical grounds, the maximum amount of cocaine, at any one sitting, should not exceed 200 mgm. The question as to the maximum amount of tetracaine or cocaine to be used topically is not easily answered. These data on absorption seem to indicate that the dictum "use the least amount of the most dilute solution to obtain effective anesthesia as possible" should be adopted.

There is a latent or lag period from the moment a drug is applied to a nerve fiber until the full effect is established. This is especially true when topical anesthetics, such as cocaine and tetracaine are being employed. As many as five, and often as long as ten minutes may elapse before full anesthesia is obtained. Clinicians are either unaware of this latent period, or become impatient and tend to ignore it and

commence surgical manipulations before anesthesia is fully established. The anesthesia is usually inadequate and a second application is often made before the first has had an opportunity to exert its effect. It can be seen how such a practice could lead to overdosage. As a rule the longer lasting drugs manifest a longer latent period.

Most physicians are generally aware of the effects of over concentration of local anesthetics on the central nervous system. Local anesthetics depress cardiac tissues; the myocardium may be so depressed that asystole results. This depressant effect of local anesthetics has been utilized to decrease prophylactically cardiac irritability and obviate arrhythmias during thoracic surgery. Local anesthetics also cause vasodilatation. Severe degrees of hypotension may result when myocardial depression and vasodilatation occur simultaneously. There is no reason to assume that both the circulatory and nervous systems cannot be involved simultaneously, particularly when overwhelming doses are used; in fact, this appears to occur in reactions from tetracaine.

Should a serious reaction occur, one is faced with two emergency situations, respiratory failure and asystole: if cardiac arrest is suspected immediate opening of the thorax and cardiac massage is indicated; if the heart has not stopped but the blood pressure is unobtainable, vasopressors, such as ephedrine, phenylephrine (neosynephrine) or methoxamine (vasoxyl) or similar drugs should be administered intravenously. Artificial respiration must be instituted immediately and maintained throughout the period of apnea or inadequate ventilation. Obviously one should not undertake the administration of local anesthetics in situations in which it is not possible to institute resuscitative measures immediately. In other words, one should have available apparatus for performing artificial respiration.

Most reactions are due to overdosage. Certain reactions, however, cannot be ascribed to overdosage, and are due to other causes. Most clinicians classify reactions as due to idiosyncrasy. In idiosyncrasy the usual therapeutic dose of drug gives a qualitative deviation from the anticipated pharmacological response; in other words a response not ordinari-

ly characteristic of a drug occurs. Idiosyncrasy is rare. Reactions characterized by hypotension, convulsions, excitement, coma, respiratory failure, etc., are not due to idiosyncrasy. These are symptoms ordinarily anticipated from overdosage. Idiosyncrasy is often confused with intolerance. Intolerance is a quantitative response. When a reaction is due to intolerance the usual response expected of the drug occurs, but with a quantity far less than the usual therapeutic dose. Intolerance also is uncommon but occurs more frequently than allergy or idiosyncrasy. It may be encountered in aged, poor risk subjects who are unable to metabolize the drug at the usual rate.

The tendency to implicate allergy as the cause of reactions is all too prevalent. Many drugs, local anesthetics included, combine with protein and become antigenic. There is a remarkable difference between symptoms caused by overdosage and by an allergic response. When the response is an allergic one, eczema, urticaria, laryngeal edema or bronchospasm develop. These symptoms, particularly those manifested by cutaneous lesions, require some time to develop. Several hours may elapse before they appear, and they persist for several days. An allergic response implies that there has been previous contact with the drug, which invariably is the case. Such sensitivity may be detected by the use of patch tests or raising intradermal wheals. It is possible for a fatal reaction to be anaphylactoid, but this is rare indeed. A minute amount of a drug applied topically intracutaneously, or by any other route, promptly causes syncope and death. The quantity ordinarily used for raising a skin wheal or for performing a conjunctival sac test may cause death. Intracutaneous or conjunctival tests are of questionable value. They may detect the occasional patient who has become sensitized and is truly allergic, but they are no value in detecting intolerance; likewise they are of no value in determining who may develop an anaphylactic response. The quantity used to perform the test would be lethal to a truly sensitive subject.

SUMMARY.

Fatalities and untoward reactions occur more frequently

when local anesthetics are applied topically than when injected. It has been assumed that rapid absorption is the cause, but direct evidence in support of this contention is scarce. The writers have determined the blood levels of tetracaine and cocaine, in dogs, after the *1.* topical application in the pyriform fossae; *2.* trachea and bronchi; *3.* after the rapid intravenous injection; *4.* after the slow intravenous infusion, and *5.* after infiltration into subcutaneous tissues.

The quantity of drug which results in no detectable blood level when infiltrated subcutaneously gives levels, when applied topically, equal to one-third to one-half of those noted after rapid intravenous injection. Epinephrine did not retard the absorption of either drug. Four per cent cocaine and 10 per cent cocaine gave similar blood levels. The basic form was as rapidly absorbed as the solution of the salt.

The absorption from mucous membranes is far more rapid than clinicians have realized and closely simulates slow intravenous injection.

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A TECHNICAL AID IN ADENOIDECTOMY.*

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Iowa City, Ia.

A great deal has been written in relation to the surgical technique of the adenoidectomy, and it is generally agreed that direct visualization of the nasopharynx is highly desirable.[1,2,3,4,5,6,7,8,9,10]

Adenoidectomy on one hand has been greatly facilitated with the development and wide acceptance of the self-retaining type of mouth gags and the use of the head-low position. The surgeon does not require assistants, and aspiration pneumonia has been reduced to a minimum. On the other hand the use of the self-retaining mouth gag and the head-low position have contributed to a poorer exposure of the nasopharynx, often resulting in incomplete adenoidectomy.

Several authors[1,7] have advocated the sitting position in preference to the head-low position for the performance of adenoidectomy. The entrance of blood into the tracheobronchial tree in this position is prevented by an endotracheal tube with an inflated cuff. The soft palate is displaced with the use of one of the different types of palate retractors available. Two assistants are required: one to move the head of the patient into different positions, and the other to keep the operative field clean by the use of suction.[1]

The dangers inherent in the sitting position under general anesthesia, we believe, have been minimized by its advocates when they state[7] that the only advantage of the head-low position is the prevention of the entrance of blood into the trachea. In other surgical fields in which the sitting position is frequently used, numerous devices have been introduced to prevent orthostatic hypotension.

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Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Dec. 12, 1957.

It is well-known that passive tilt to the sitting position can be followed by hypotension even in normal subjects. If the compensatory reflexes are impaired by the commonly used pre-anesthetic medications the postural hypotension is greatly increased.[12] The hypotension may be followed by cerebro-vascular accidents. We believe that this technique has definite advantages, but should not be used routinely because of the inherent dangers. In cases of secondary adenoidectomies when there is a great deal of tension of the soft palate as a result of scarring, the erect position may be the procedure of choice and the only way in which a complete adenoidectomy can be performed.

If an adenoidectomy is to be performed under direct visualization the self-retaining type of mouth gags should not be employed. since they produce tension of the soft palate. This makes exposure of the nasopharynx extremely difficult, and under these circumstances if the operator uses a palate retractor, lacerations of the soft palate will frequently occur.

In our primary adenotonsillectomy we routinely use a self-retaining mouth gag and the head-low position. A No. 4 laryngeal mirror (see Fig. 1) that has been previously immersed in a glass containing warm saline is placed in the oropharynx of the patient for indirect visualization of the nasopharynx.

For the removal of lymphoid tissue from the nasopharynx various instruments can be used according to the needs of the individual case. We prefer the Barnhill and Jones curettes,[11] the cutting edges of which should be kept sharp at all times. The Meltzer punch forceps are especially useful for the removal of smaller collections of lymphoid tissue in the fossae of Rosenmueller. The shanks of these instruments help in the retraction of the soft palate. With one hand the laryngeal mirror is manipulated and with the other the cutting instrument.

The main mass of midline adenoid tissue is removed with the first two or three strokes of the curettes. The nasopharynx is kept free of blood with the Yankauer suction tube. If bleeding interferes with further work in the nasopharynx

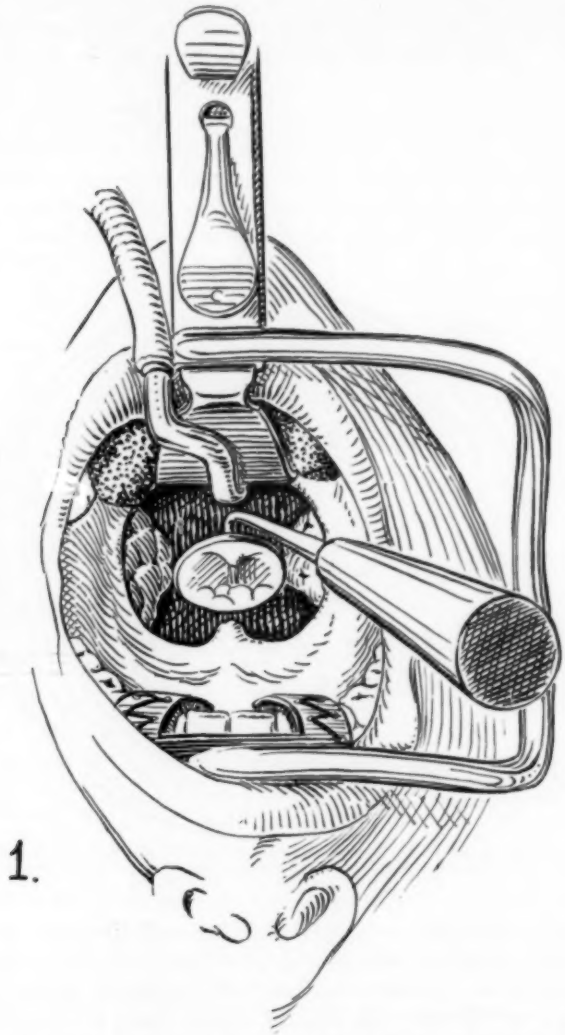

Fig. 1. A laryngeal mirror previously warmed in hot saline is placed in the oropharynx of the patient for indirect visualization of the nasopharynx.

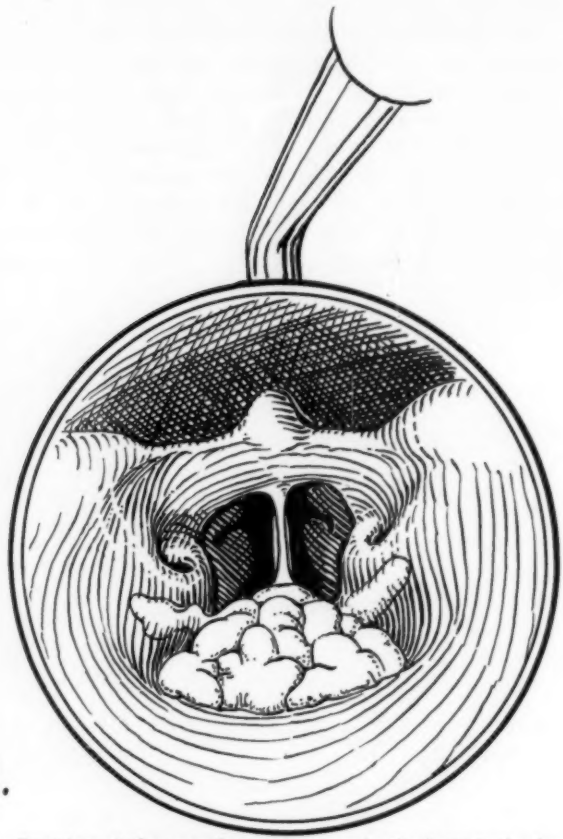

Fig. 2. Drawing of the nasopharynx as seen in the laryngeal mirror. The soft palate is not shown in these figures.

a post nasal pack is inserted, and the tonsillectomy is performed. After its completion, attention once more may be directed to the nasopharynx, and any remnants of lymphoid tissue removed with the Meltzer punch forceps.

We have been using this technique for the past five years with very gratifying results. The view obtained by the indirect method (see Fig. 2) is inverted from that obtained

by direct method.[2] Because of the inverted image obtained the surgeon tends at first to make motions in reverse, but after the first few cases this initial difficulty is easily overcome.

Caution should be taken when removing lymphoid tissue from the fossae of Rosenmueller with a curette or sharp dissection. The mucosa of the medial side of the Eustachian tube should be left undisturbed otherwise adhesions will result from the healing of two opposite raw areas[9] (see Figs. 3a and 3b).

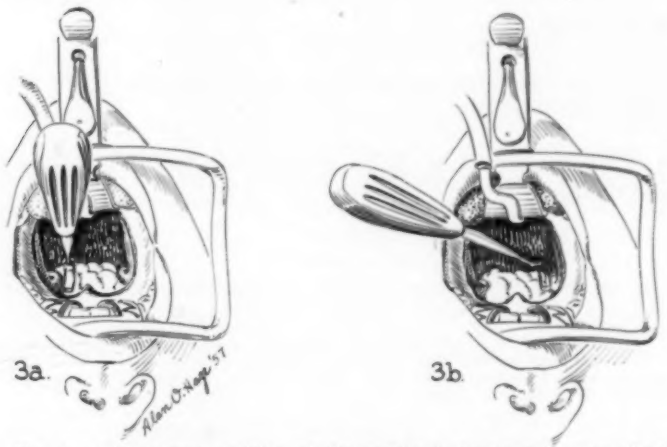

Fig. 3a. The correct position of the curette is illustrated. The shank of the instrument is placed vertically, avoiding the Eustachian tube.
Fig. 3b. The curette is improperly placed. If it is placed obliquely the posterior aspect of the Eustachian tube may be lacerated.

A review of the literature showed that one author[13] had described the technique we have advocated. A recent publication[14] also outlines a similar procedure. The writer emphasizes the removal of the highest portion of the adenoids located immediately posterior to the choanae with a Takahaski forceps introduced through the nose and visualized with the laryngeal mirror placed in the oropharynx. We have also practiced this procedure on several occasions, with satisfactory results.

The adenoidectomy should not be performed blindly or by palpation. These methods should be delegated to the historical background of the operation, but its use in modern surgery should be condemned.

SUMMARY.

1. The sitting position should not be used routinely in the performance of the adenoidectomy because of orthostatic hypotension and possibly cerebro-vascular accidents.

2. A technique of indirect visualization of the nasopharynx is described.

3. This technique has been used in the past five years with encouraging results.

4. Blind methods in the performance of the adenoidectomy are condemned.

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OTOLARYNGIC RADIOLOGY COURSE.

The Departments of Radiology and Otolaryngology, College of Medicine, the Ohio State University, will offer a post-graduate course in otolaryngic radiology February 4 and 5, 1958, at University Hospital, the Ohio State University Health Center, Columbus, Ohio, under the direction of John F. Holt, M.D., Professor of Radiology, University of Michigan; Lewis Etter, M.D., Professor of Radiology, University of Pittsburgh; Roderick Tondreau, M.D., Professor of Radiology, University of Pennsylvania; Sidney Nelson, M.D., Professor of Radiology, the Ohio State University.

There will be lecture periods with time given for group discussion. Dr. Holt will discuss the esophagus, salivary glands, tracheo-bronchial tree, and skull proper. Dr. Etter will discuss the paranasal sinuses, nose and nasopharynx. Dr. Tondreau will discuss the mastoid and larynx.

Registrants are encouraged to bring interesting roentgenograms for participation in discussion periods.

A fee of $35.00 must accompany the registration application. Luncheon for the two days will be included in this fee. All registrations are to be completed by January 15, 1958.

Address applications or requests for additional information to: William H. Saunders, M.D., Department of Otolaryngology, University Hospital, Columbus 10, Ohio.

BOOK REVIEWS.

Sprachaudiometrie Grundlagen und Praktische Anwendung Einer Sprachaudiometrie für das Deutsche Sprachgebiet. Doz. Dr. Med. Karl-Heinz Hahlbrock. Georg Thieme Verlag, Stuttgart (in the U.S.A. and Canada, Intercontinental Medical Book Corporation, New York 16, N. Y. 1957. Soft cover, 182 pp. $5.00.

Dr. Hahlbrock has produced a comprehensive systhesis of the principles and practices relating to speech audiometry. The chapters include the development of speech audiometry; the practical procedures, interpretation of test results; comparisons between classical tests of hearing and speech audiometry and the techniques of application of speech audiometry to the selection of patients for surgery and the selection of hearing aids.

For those who can understand the German language this should be a useful guide to speech audiometry. There is a short introduction by Prof. Dr. F. Zöllner.

Educational Guidance and the Deaf Child. Edited by A. W. G. Ewing. The Volta Bureau, 1537 35th St. N.W., Washington 7, D. C. 1957. 345 pp.

A. W. G. Ewing, Professor and Director of the Department of Education of the Deaf, University of Manchester, has brought together a collection of studies that reflect the activity in the Department of Education of the Deaf at his University. The major portions of the volume deal with infancy and early childhood and children of school age. A good deal of emphasis is placed on screening tests and guidance for parents, and on tests of abilities and attainments. The book is a useful compendium for those who want to acquaint themselves with the forward-looking activities at Manchester.

Look, Listen and Lipread. Edited by Betty C. Wright. The Volta Bureau, 1537 35th St. N.W., Washington 7, D. C. 1957. Soft cover, 110 pp.

Miss Wright, who is listed as Consultant, American Hearing Society and U. S. Office of Vocational Rehabilitation; Instructor of Lipreading, Hearing Center, Medical College of Virginia, has gathered together contributions from experienced teachers of lipreading that the latter have found useful in practice. The compendium is a useful source of tried material for the teacher of lipreading.

S. R. S.

SUBCOMMITTEE ON HEARING IN CHILDREN.

The American Academy of Ophthalmology and Otolaryngology, through its Subcommittee on Hearing in Children of the Committee on Conservation of Hearing, has been conducting a long-term nationwide study of problems relating to the conservation of hearing in children. The specific aims are to develop the most efficient case-finding methods and to use these methods in estimating the magnitude of the problem in the country; to study state laws and review current practices and facilities for rehabilitation of hearing impaired children; to help develop methods for medical and surgical rehabilitation standards; and ultimately to use the Subcommittee findings in assisting professional workers to improve and enhance programs in hearing loss.

In the second year of operations, a full-time Executive Director has been engaged, and offices established at the Graduate School of Public Health, University of Pittsburgh. An initial study is being conducted in Pittsburgh to identify early medical signs and symptoms which may indicate danger of hearing impairment, to measure the psychological, social and other effects of such impairment and to develop efficient and economical methods for the testing of hearing in children. The Pittsburgh study is a cooperative effort among the following: The Subcommittee on Hearing in Children, the Graduate School of Public Health and the School of Medicine of the University of Pittsburgh, the Pittsburgh Board of Public Education, and the Allegheny County Department of Health.

The members of the Subcommittee on Hearing in Children are: Dr. John E. Bordley, Baltimore; Dr. Victor Goodhill, Los Angeles; Dr. Hollie E. McHugh, Montreal; Dr. S. Richard Silverman, St. Louis; and Dr. Raymond E. Jordan, (Chairman) Pittsburgh. An advisory committee of consultants from the University of Pittsburgh includes Dr. Samuel M. Wishik, Dr. Leo G. Doerfler, and Dr. Isidore Altman. Grants from the United States Children's Bureau through the Pennsylvania Department of Health and from the National Institutes of Health are providing financial support.

THE INTERNATIONAL CONGRESS ON THE EDUCATIONAL TREATMENT OF DEAFNESS.

The University of Manchester Department of Education of the Deaf, will sponsor The International Congress on the Educational Treatment of Deafness, July 15-23, 1958.

The Minister of Education for England and Wales has decided to recognize the Congress as a Short Course which teachers of the deaf may attend under Grant Regulations.

An interesting program has been arranged.

Demonstrations will be given to illustrate some aspects of the research that is in progress in the audiology unit and audiological laboratories of the Department of Education of the Deaf, and in collaboration with the Department of Otolaryngology, the Public Health Department and School Medical Service of the City of Manchester, and the Ear, Nose and Throat Department of the Royal Manchester Children's Hospital. The subjects of the demonstrations will include procedures for the making of screening tests and assessments of the hearing of babies and young children, the results of guidance to parents to enable them to give home training to their children of pre-school age whose hearing is impaired, and the results of an experimental investigation on problems in architectural acoustics that concern the use of hearing aids in school buildings.

Arrangements are being made to offer accommodation to members of the Congress in University Halls of Residence. The cost of this type of accommodation is expected to be about £1. 2s. od. per day, including bed, breakfast and dinner. The University refectory will be available for meals during each day.

For those members who wish to do so, arrangements will be made to visit audiology clinics and special schools in London, Oxford and other places. In connection with the Congress it is hoped to organize tours to places of interest. The dates proposed for this are July 9th, 10th, and 11th.

For further details write: Prof. A. W. G. Ewing, Department of Education of the Deaf, The University, Manchester 13, England.

THE 11TH CONGRESS OF THE INTERNATIONAL ASSOCIATION OF LOGOPEDICS AND PHONIATRICS.

The 11th Congress of the International Association of Logopedics and Phoniatrics will take place in London, August 17-22, 1959. The following official reports will be presented: The Inheritance of Voice and Speech Disorders, Prof. R. Luchsinger, M.D., Zürich; Defects of Articulation, Muriel Morley, B.Sc., F.C.S.T., Newcastle; The Physiology and Pathology of the Soft Palate, Prof. Lucio Croatto, M.D., Padua. The official languages of the Conference will be: English, French, and German.

Those working in the field of Speech and Voice Therapy and all who are interested in this specialty are invited to attend and to submit papers.

If intending to be present, whether submitting a paper or not, please inform the Congress Secretary at the earliest possible date.

Papers are invited on subjects relevant to the three main reports or any other aspect of speech and voice. Only one contribution will be accepted from any one member of the Congress and this must not have been previously published. The Committee reserves the right to select papers. Papers should be restricted to 15 minutes in length; demonstrations to 10 minutes. In special cases 20 minutes may be allowed for a paper of great importance but only if application is made at the time of submitting the title. Should a group of four or more people wish to present a symposium or a prepared discussion on a theme, consideration will be given to allocating up to one hour for such a contribution.

Titles must be received by September, 1958. Summaries must be received not later than November 15th, 1958.

All communications are to be sent to: Peggy Carter, L.C.S.T., 46 Canonbury Square, London, N. 1.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill.

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Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Square, Philadelphia, Pa.
Treasurer: Dr. Verling K. Hart, 106 W. 7th St., Charlotte, N. C.
Meeting: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3, Pa.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: Fairmont Hotel, San Francisco, Calif., May 19-20, 1958.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14, Minn.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco, Calif., May 21-23, 1958.
Place: The Homstead, Hot Springs, Va., March, 1959.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

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Alternate Delegate: Dean Lierle, M.D., Iowa City, Iowa.

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Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14, Minn.
Place: Fairmont Hotel, San Francisco, Calif., May 17-18, 1958.
Place: The Homestead, Hot Springs, Va., 1959.

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Annual Meeting: October, 1958, Chicago, Ill. (Definite time and place to be announced later).

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Irvin J. Fine, 506 New Brunswick Ave., Perth Amboy, N. J.
3Secretary: Dr. Samuel M. Bloom, 123 East 83rd St., New York 28, N. Y.
Meeting: Columbus, Ohio, March 7-8, 1958.

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Meeting quarterly (March, May, October and December), on the second Thursday of the month, 6:30 P.M. at Urmey Hotel, Miami.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

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KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

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LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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Chairman of Ophthalmology Section: Dr. Richard Kratz.
Secretary of Ophthalmology Section: Dr. Carrall A. McCoy.
Chairman of Otolaryngology Section: Dr. Howard G. Gottschalk.
Secretary of Otolaryngology Section: Dr. Robert W. Godwin.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P. M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

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Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
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